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**ДЕТСКАЯ АНЕСТЕЗИОЛОГИЯ**  
**И**  
**РЕАНИМАТОЛОГИЯ**

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Москва «Медицина»

VA MIKHELSON

ANAESTHESIA  
AND  
INTENSIVE THERAPY  
FOR CHILDREN



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## Preface

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Anaesthesiology and intensive therapy are branches of medicine that are very intimately connected with other medical specialities such as surgery, therapy, obstetrics, gynaecology, paediatrics, etc., because the anaesthesiologist has to provide anaesthesia and intensive therapy to patients with various diseases. The anaesthesiologist uses strong medicines and intensive methods to act on the human body. This requires profound knowledge of clinical biochemistry, pharmacology and some laws of physics. Moreover, the anaesthesiologist must be able to handle complicated instruments and apparatus and hence his engineering skill and knowledge must be perfect too. The anaesthesiologist must be well prepared to meet many problems other than purely anaesthesiological and requiring knowledge in other related branches of medicine for their solution.

Paediatric anaesthesiology is part of general anaesthesiology and intensive therapy. The principles of paediatric anaesthesiology are the same as of anaesthesiology for adults. The main task of an anaesthesiologist is to protect the patient from operational stress and to control and maintain the vital functions of the patient in critical conditions. But the methods that are applied to infants and children can differ significantly from those used for adult patients. Neonates and infants require special care. The special anatomy and physiology of older children also account for special care of these patients.

The textbook includes three parts. The first part deals with the general problems in anaesthesiology, intensive therapy and resuscitation, and also organization of the anaesthesiological and intensive therapy service (Plate 1), the apparatus and medicines used for anaesthesia and intensive therapy. It also discusses the anatomical and physiological properties of children from the viewpoint of the anaesthesiologist.

The second part is dedicated to clinical anaesthesiology in paediatric practice. This part deals with the general principles and methods of anaesthesia in children, the problem of selection of anaesthetic methods and techniques depending on the child's condition and the character of surgery.

The third part deals with clinical resuscitation and intensive therapy. Special emphasis is laid on correction and maintenance of vital functions in children with common diseases. Intensive therapy of children with various diseases is described.

This textbook is the result of many years' intensive work of the author and his colleagues in the field of paediatric anaesthesiology and intensive therapy at Filatov's Paediatric Clinic in Moscow.



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## PART ONE

# General Problems of Paediatric Anaesthesiology and Intensive Therapy

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### Chapter 1

## Paediatric Anaesthesiology and Intensive Therapy

Anaesthesiology and intensive therapy, resuscitation included, are very important aspects of modern paediatric medicine. Their importance depends on the problems and principles that determine the essence of anaesthesiology and intensive therapy themselves. They obey the same laws that govern the progress of general anaesthesiology and intensive therapy, it would also be incorrect to regard paediatric anaesthesiology and intensive therapy as minor anaesthesiology and intensive therapy. They are the same medicine but only adapted to children. In some cases the little patient requires even more extensive medical care than an adult, because of the special features of his physiology and anatomy and the special character of children's pathologies. All branches of paediatric medicine therefore are specific. This concerns deontology, selection of methods for anaesthesia, intensive therapy and resuscitation, indications for various medicines and their dosage, and many other aspects. It is quite evident that a paediatric anaesthesiologist must have profound knowledge in paediatrics. Love for his patient and the desire to lessen his sufferings is another prerequisite condition for a paediatric anaesthesiologist.

*Anaesthesiology* is a branch of clinical medicine, whose main object is to protect the patient from surgical stress. An operative intervention is a surgical correction of various dysfunctions, but the body reacts to any operation or painful manipulation. The degree of stress depends on the psychic trauma, pain syndrome, and homeostatic changes that occur during operation (blood loss, upset gas exchange, biochemical shifts, etc.). The reaction of the human body to an operation is quite varied. Incision of tissues, loss of blood, opening of the chest or abdomen, manipulations on reflexogenic zones, etc., can be the triggering mechanism of this reaction. They can cause respiratory distress, circulatory disorders, changes in the blood coagulating and anti-coagulating system, and metabolic disorders. The neurovegetative system responds by spasms in the peripheral vessels, release of excess catecholamines into the blood,

and by various metabolic disorders. A vicious circle is thus formed and many homeostatic disorders become independent of their provoking factors and they themselves become the cause of further changes in the body. The task of an anaesthesiologist is to remove pain, render a patient unconscious during operation, and to ensure safety of the child during operation and in the early post-anaesthesia and post-operative periods. In other words, gas exchange, haemodynamics and other homeostatic parameters of the child should be maintained within safe range.

*Intensive therapy and resuscitation* deal with terminal states of the body, functioning and methods of their treatment. But this is not the main and the only object of intensive therapy. As this branch of medicine has been developing, it proved to be effective not only for terminal states, but also for cases with upset vital functions. It is quite natural that the number of such cases is much higher and intensive therapy is now regarded as a means of prevention of terminal states.

The range of application of resuscitation measures has now broadened greatly. The problems of intensive therapy can be formulated as follows: (a) the study of pathophysiological processes occurring during the terminal state and in severe (critical) conditions, (b) treatment of patients in the terminal state (resuscitation or revival), (c) treatment of patients with severe vital dysfunctions (severe or critical condition).

**Terminology.** We shall only discuss several terms that are of major importance for the determination of the main concepts in anaesthesiology and intensive therapy. Other terms will be defined later in appropriate chapters.

*Narcosis* (Gk *narkosis*, a numbing) is a depression of the central nervous system by special substances (narcotics or anaesthetics) characterized by stupor, insensibility (absence of consciousness or sensitivity to pain or other stimuli) and reflex depression. This condition is reversible and the initial status is restored after suspension of administration of the drug. The term is close in its sense to 'anaesthesia', which is however less accurate because 'anaesthesia' does not imply loss of consciousness, only 'general anaesthesia' implies unconsciousness.

*Anaesthesia* (an neg + Gk *aisthesis*, sensation) is loss of feeling or sensation induced by drugs. 'Local anaesthesia' is commonly used to mean anaesthesia confined to one part of the body.

*Resuscitation* (L *resuscitare*, to revive) means restoration of vital functions in patients in the terminal state, or apparently dead patients. The term 'cardiopulmonary resuscitation' is confined to re-establishment of the heart and lung function, but does not imply the whole complex of problems that a physician has to manage for the resuscitation (revival) of patients. Artificial maintenance of blood

circulation and respiration re-establishes the vital functions of the body

*Intensive therapy* is the treatment of patients in whom one or several vital functions are so disordered that the patient cannot live longer unless these dysfunctions are compensated artificially. Acute disorders are mainly understood in such cases (Plate 2)

Intensive therapy is always compensatory in its character and is intended to replace artificially a completely or partly lost function, as for example artificial ventilation of the lungs, parenteral nutrition, haemodialysis, bronchoscopic sanitation (to maintain airway patency), etc. Another special feature of intensive therapy is that it is often directed to eliminate a syndrome. Resuscitation means aid to patients in whom the diagnosis may not be accurate, while the necessity of a pathogenetic therapy is quite obvious. The clinical picture in such cases is characterized by the domination of one or several syndromes that should be quickly corrected to preclude death of the child. Such cases include acute respiratory distress, metabolic acidosis or alkalosis, acute renal failure, shock, hyperthermia, convulsive syndrome, etc. The physician should therefore start with treatment of syndromes and then proceed to pathogenetic therapy. It is quite natural that in some cases the therapy of syndromes is actually a pathogenetic therapy.

A severe syndrome develops usually when a vicious circle in the genesis of a disease is formed. For example, acute respiratory failure associated with severe forms of stenosed laryngitis (croup) develops secondary to viral infection, which is followed by obstruction of the upper airways, hypoxia, hypercapnia, and anxiety. This, in turn, increases the oxygen demand and the release of catecholamines and intensifies inflammation. Treatment of only hypoxia or hypercapnia will not be efficient in such cases, because treatment should be aimed at elimination of inflammation, control of infection, etc. The third specific feature of intensive therapy is thus a complex therapy of all links in the pathological circle characterizing a given severe syndrome.

*Intensive control* implies permanent monitoring of the patient's condition. Intensive control should be given to children recovered from critical conditions, but in whom the function of the vital organs and systems can at any moment be impaired again. Children with acute poisoning, newborns and premature infants also need intensive control.

## Chapter 2

# Equipment and Apparatus Used for Anaesthesia, Intensive Therapy and Resuscitation of Infants and Children

### ANAESTHETIC APPARATUS

An anaesthetic apparatus is used (a) to supply gaseous or volatile anaesthetics, oxygen or air into the patient's airways at a preset flow rate, (b) to maintain the needed temperature and humidity of the inhaled gas, (c) to remove exhaled gas from the airways, (d) to eliminate carbon dioxide from the exhaled gas, and (e) to ensure assisted or artificial ventilation of the patient's lungs

Anaesthetic apparatus (Fig 1) usually includes the following three main units (1) reservoirs (cylinders) for compressed oxygen and gaseous anaesthetics (with reducing valves), (2) vaporizers for liquid anaesthetics provided with flow-meters for gaseous anaesthetics oxygen and air, (3) breathing circuits for circulation of the narcotic mixture

A simple vaporizer works as follows. An anaesthetic supplied from a flow-meter may (a) by-pass the vaporizer, the concentration of the anaesthetic in the breathing gas is then zero, (b) the anaesthetic can be partly delivered into the vaporizer and its vapour will then be mixed with other gaseous components at the outlet, (c) the entire quantity of anaesthetic may pass through the vaporizer to ensure the maximum concentration of the anaesthetic in the breathing gas

There are semiautomatic and automatic systems in which the effect of varying temperature on evaporation of the anaesthetic can be compensated to ensure its constant concentration in the breathing mixture

The anaesthetic system includes also absorbing device in which carbon dioxide exhaled by the patient is absorbed (in rebreathing circuits). In closed or semi-closed systems, carbon dioxide is returned into the anaesthetic apparatus, and if the supply of fresh gas is insufficient, carbon dioxide concentration can increase to a dangerous level. A  $\text{CO}_2$  absorber eliminates this danger

Various *valves* are used in anaesthetic apparatus. Some of them (one-way or non-rebreathing valves) are used to ensure flow of gas in one direction only, while others are intended to release excess pressure in the system or to vent exhaled gas to atmosphere. A non-rebreathing (one-way) valve is used to separate the flows of inhaled and exhaled gases during both spontaneous and artificial ventilation of the lungs

The system includes also connecting hoses and tubes, adapters, tapers, endotracheal tubes, a face mask, and a reservoir bag

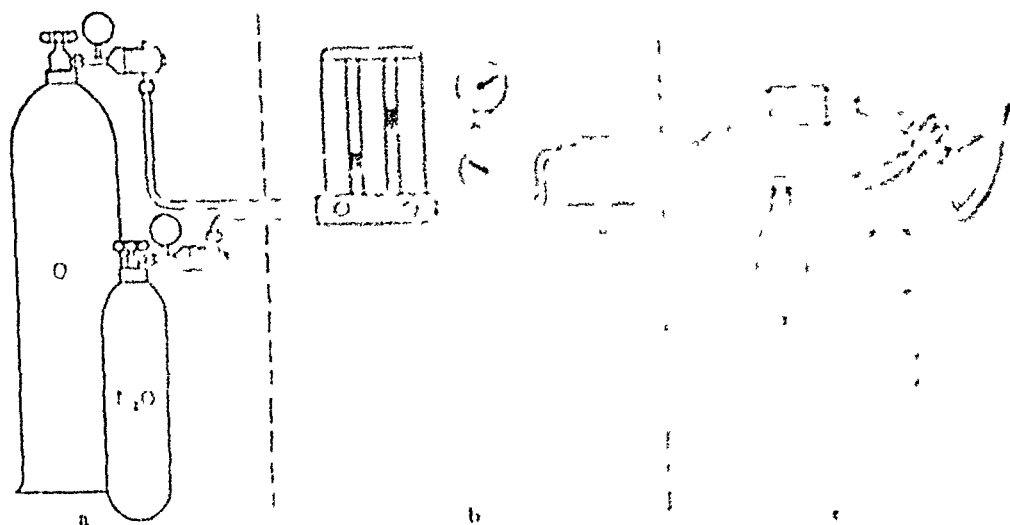


Fig. 1 Anaesthetic apparatus

a—compressed gas cylinder    b—flowmeter    c—patient

Absorption and non-rebreathing systems are distinguished by the *absorption system* part or the entire exhaled gas is returned to the anaesthetic apparatus for repeated inhalation. Carbon dioxide absorbers remove  $\text{CO}_2$  from the exhaled gas. The dead-space effect in this apparatus is insignificant. The advantage of this system are economy of oxygen and the anaesthetic, in remittance loss of heat and moisture by the patient, low breathing resistance, and decreased atmospheric pollution in the operating theatre.

The disadvantages of the absorption system are difficult control of the anaesthetic concentration in the exhaled gas and difficult disinfection of the apparatus.

Absorption systems can be *circular* or *to-and-fro*. In the circular absorption system, gases pass from the apparatus to the patient and back to the apparatus (Fig. 2a). Part of the exhaled gas can be discharged to atmosphere through a half-open exhale valve or through a release valve of the apparatus. The amount of discharge from the system depends on the rate of fresh gas flow: the higher the fresh gas flow to the breathing system, the larger the discharge of the exhaled gas. The exhaled gas passes an absorber where carbon dioxide is retained.

With the *to-and-fro* system (Fig. 2b), the inhaled and exhaled gases move alternately through the same hose (from the apparatus to the patient, and back). The exhale valve can be either closed or half-open.

In an absorption system all exhaled gas is returned to the apparatus. If part of exhaled gas is vented to atmosphere, the circuit is called *semi-closed*.

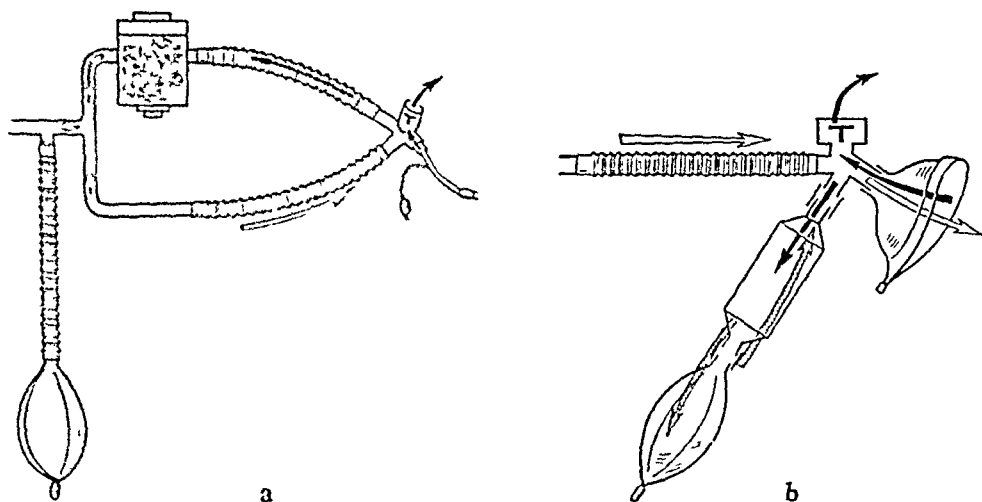


Fig 2 Breathing systems

a—circle system, b—to-and-fro system

In a *non-rebreathing system* all exhaled gas is vented to atmosphere through a special exhale valve. The inhale valve prevents the return of the exhaled gas back into the apparatus, and this makes the  $\text{CO}_2$  absorber unnecessary. But the  $\text{CO}_2$  content in the inhaled gas may be higher than in atmospheric air due to the dead-space effect, inadequate function of the inhale valve, or excessive volumes of various elements of the system (connectors, T-pieces, etc.). Since the exhaled gas is not rebreathed, the anaesthetic concentration in the fresh gas flow should be higher to ensure the required effect.

A non-rebreathing system can be either open or semi-closed. With the open circuit, the anaesthetic vapour is inhaled from and exhaled into atmosphere. A typical example of an open circuit is inhalation of an anaesthetic which is dropped on a gauze-covered Esmarch mask. When a child breathes spontaneously, he inhales the anaesthetic vapour together with atmospheric air. The method is now used only in rare cases and is absolutely prohibited for use with neonates because of the great danger of overdosage.

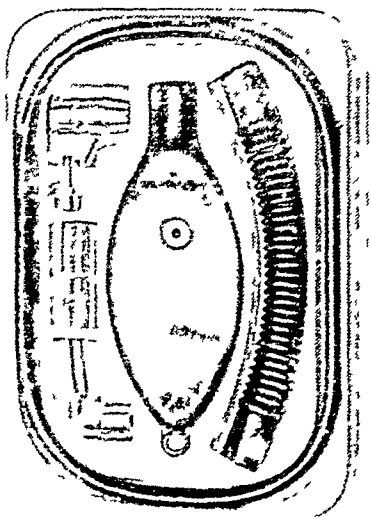
With a semi-closed circuit, an anaesthetic mixture from the cylinder passes a flow-meter and a vaporizer before it is inhaled. Exhaled gas is discharged to atmosphere. Both liquid and gaseous anaesthetics can be used in mixtures with oxygen. Special care should be exerted when giving anaesthesia to infants under 6 years of age. Non-rebreathing valves with semi-open circuits should be used with neonates. Since valvular systems have their disadvantages, they should not be used with neonates and infants whenever possible. Valveless systems should be preferred.





Fig 3 Conducting anaesthesia using an Ayre (T-piece) system

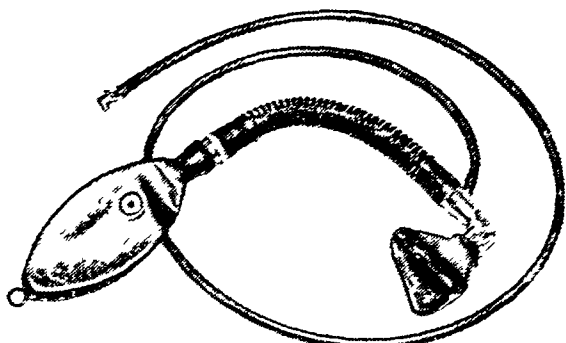
The Ayre system (Fig 3) is a T-piece with a diameter of 1.5 cm. Fresh gas flow passes through a hose fixed to one limb of the T-piece, while the other limb is connected to the endotracheal tube. The third limb remains open to discharge the exhaled gas. This limb is closed by a finger during inhalation and the child therefore breathes in fresh gaseous anaesthetic. During exhalation the pressure of the



a

Fig 4 An anaesthetic attachment for newborns and infants

a—packed in a kit, b—assembled for use



b

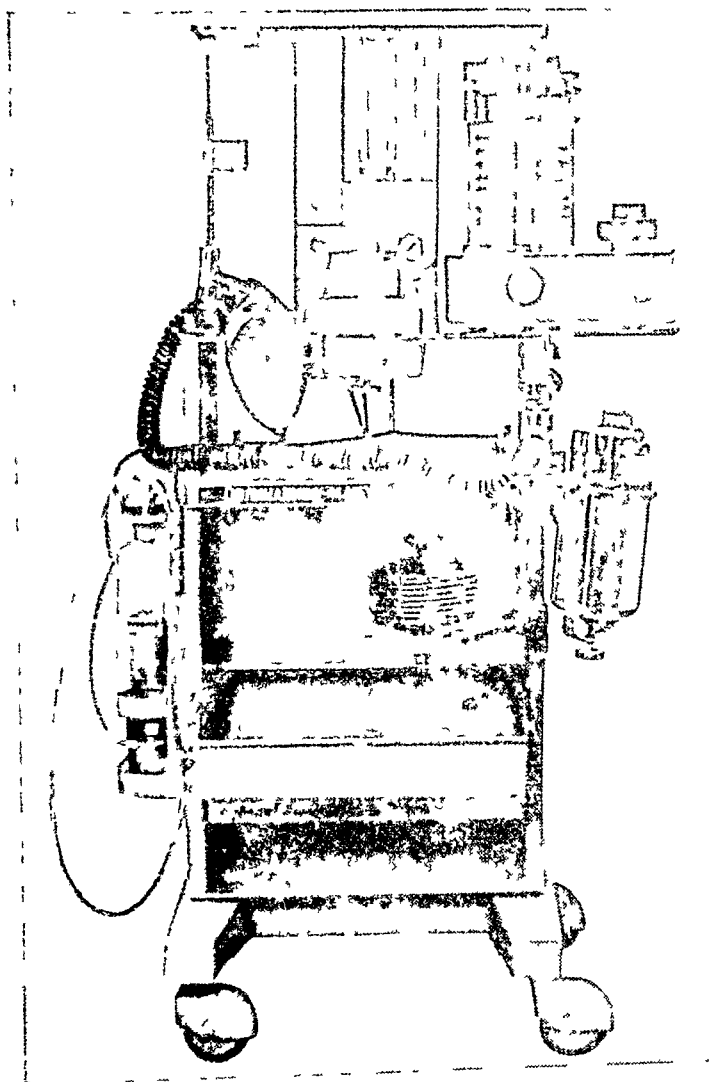


Fig 5 Soviet-made all-purpose anaesthesia apparatus 'Polynaicon-4'

finger should be released. In order to prevent undue loss of gases and to prevent suction of atmospheric air, a rebreathing tube is attached to the expiratory limb. The length of the tube (and, hence, its volume) varies depending on the age of the child. The fresh gas flow should be 2.5 times higher than minute ventilation rate. This is necessary to prevent hypercapnia. (For neonates fresh gas flow should be 4-5 l/min.)

Any anaesthesia apparatus intended for adults can be used for inhalation anaesthesia of children older than 3 or 5. The selection of the apparatus depends on the child's condition, duration of

anaesthetic sleep, the amount of operative injury and the character of operation, and also on the skill of the anaesthesiologist. The main requirements for the anaesthesia apparatus for small children are as follows: (a) minimum possible breathing resistance (especially expiration resistance), (b) minimum dead space, (c) maintenance of temperature and humidity of inhaled mixture within normal range. These requirements are met with a special anaesthetic attachment shown in Fig. 4 or with absorption systems in which a significant portion of the exhaled gas is vented to atmosphere.

The Soviet-made apparatus 'Polynarcon-4' (Fig. 5) is a very convenient all-purpose apparatus.

### APPARATUSES FOR ARTIFICIAL LUNG VENTILATION

Ventilators are used for intermittent delivery of breathing gas into the lungs for their ventilation. A great variety of apparatuses for artificial ventilation of the lungs are used in paediatric anaesthesiology and intensive therapy. Their operating principle is based on mandatory air delivery into the lungs. The energy which is necessary to perform this work is either compressed gas, electricity, or muscular power. In accordance with the mode in which the inhalation and exhalation processes are alternated, the ventilators are divided into three groups. Their action is thus controlled by *pressure* inside the breathing circuit, by *volume* (by amounts of delivered gas), and by *frequency* (by the timer). Practically any inhalation anaesthetic apparatus can be used for manual ventilation of the lungs. A reservoir bag is used for the purpose. As such a bag is compressed, the gas mixture is expressed into the airways of the child, exhaled gas is discharged to atmosphere.

A Soviet-made all-purpose ventilator 'RO-6' (Fig. 6) is intended for artificial ventilation of the lungs (both controlled and assisted), for induction of anaesthesia, and during surgical operations or resuscitation procedure. The inhalation is active while exhalation is passive. Any anaesthetic preparation can be given to the patient with this apparatus. The ventilator is provided with a sucker (operated by compressed gas), a volumeter, and a humidifier. Assisted respiration helps the patient to re-establish his spontaneous respiration. The preset ratings are maintained automatically. Assisted ventilation can be maintained manually using any type of breathing system.

Adult ventilators can be used with children as well, but the respiratory and minute volumes of the lungs should be accurately measured, and the resistance to respiration (first of all to expiration) should be decreased. This can be attained with a semi-closed system and non-rebreathing valves. Artificial ventilation can also be conducted with alternation of positive and negative pressure. This helps

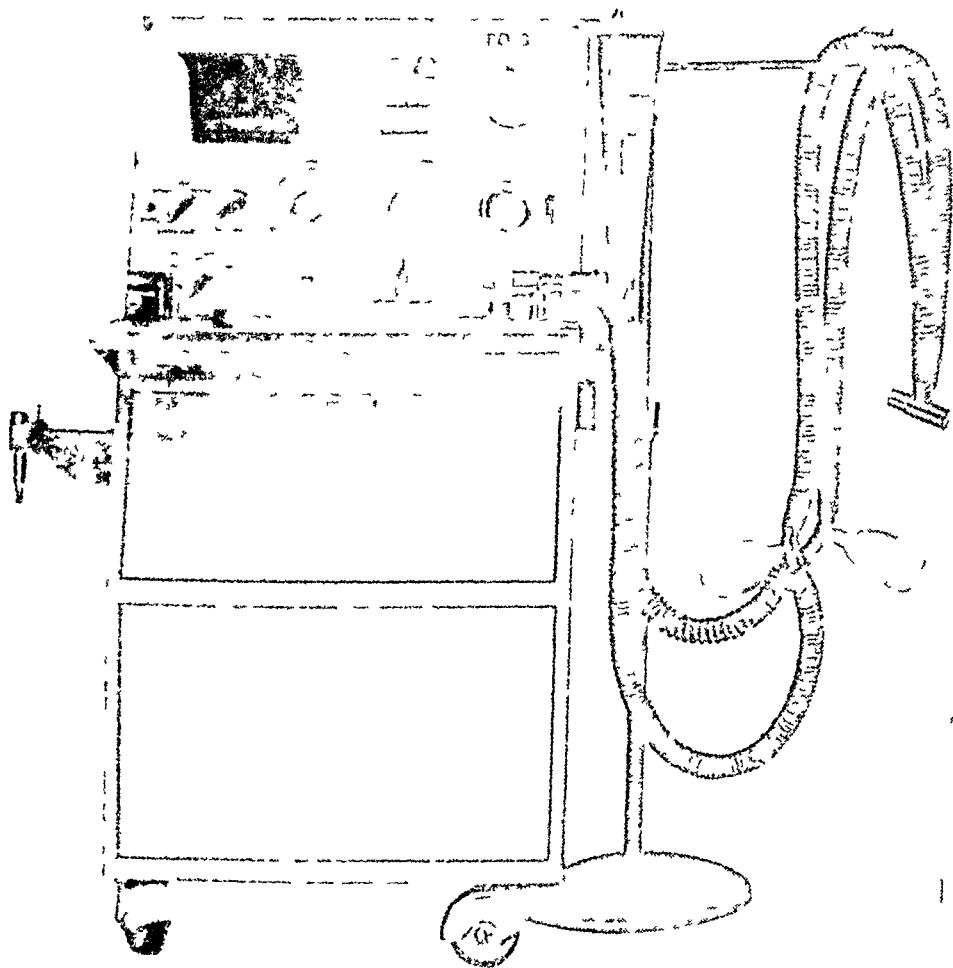


Fig 6 All-purpose apparatus for artificial lung ventilation (RO-6)

to overcome the expiratory resistance. Dead space of the apparatus is reduced to the minimum by using a non-rebreathing valve and by decreasing the volumes of the non-separated portion of the breathing system, adapters, connectors, etc.

Dead-space volume should not exceed 3 ml for a neonate, 10 ml for a 1-year-old infant, and 15 ml for a 6-year-old child. It should be remembered that normal gas exchange in a child can be maintained only if his tidal volume is at least ten times greater than the dead space of the apparatus.

There are many Soviet-made and foreign apparatus which ensure adequate ventilation of the lungs within a very wide range of tidal volumes, breathing rates, and inhalation-to-exhalation ratios.

## SAFETY REGULATIONS

Substances used for anaesthesia are inflammable. Therefore there is compressed gas cylinders. In order to prevent explosion or fire, safety regulations should be strictly followed.

Safe conduct in the operating theatre. Faulty electrical equipment that can produce sparks must not be used in anaesthesia rooms. All electrical equipment, which is indispensable for anaesthetic procedures, should be spark-proof. Control points mounting electric plugs should be installed not lower than 1.6 m from the floor level. All connectors, electrical plug and wire should be intact. Use of open flame (alcohol burners and the like) in the operating room is prohibited. If electric coagulators are used during operation, anaesthesia should be done by mixtures of oxygen with nitrous oxide, halothane or other non-flammable substances. Resistance to earth and any point of the circuit should not exceed 1 ohm. Instruments of the personnel should be made of material that do not accumulate static electricity on them. Wool, silk or synthetic garments are thus excluded. The patient and the personnel should wear only cotton clothes, the footwear (soles) of the personnel should be leather or antistatic rubber. Wearing watches, ring or other metal objects is prohibited. Relative humidity of air in the room should be not less than 60 per cent. The operating and anaesthesia room should be provided with plenum-exhaust ventilation ensuring the motion of air in the direction from the legs to the head of the patient. The exhaust opening should be not higher than 60 cm from the floor level because vapours of ether and cyclopropane are heavier than air. Fresh air should be delivered from top. The renewal rate should be 6 per hour. Compressed gas cylinder should be handled with care to prevent knocks. A special wrench should be used to open the cylinder. Never strike the valve with the wrench! The cylinders should be placed away from heating appliances. It is prohibited to pump gases from one cylinder to another.

### TOOLS AND FITTINGS USED FOR ANAESTHESIA AND ARTIFICIAL LUNG VENTILATION

Various fittings and devices are used to ensure tightness between the apparatus and the airways of the patient. These devices include face masks, endotracheal tubes, tracheostomy tubes, connectors, corrugated hoses and tubes, rubber tubes, reservoir bags, and others.

*Connectors* (adapters) are elements that connect the anaesthesia apparatus and the endotracheal tube. They may have various diameters and shapes. Curved, rather than L- or T-shaped connectors, should preferably be used with children.

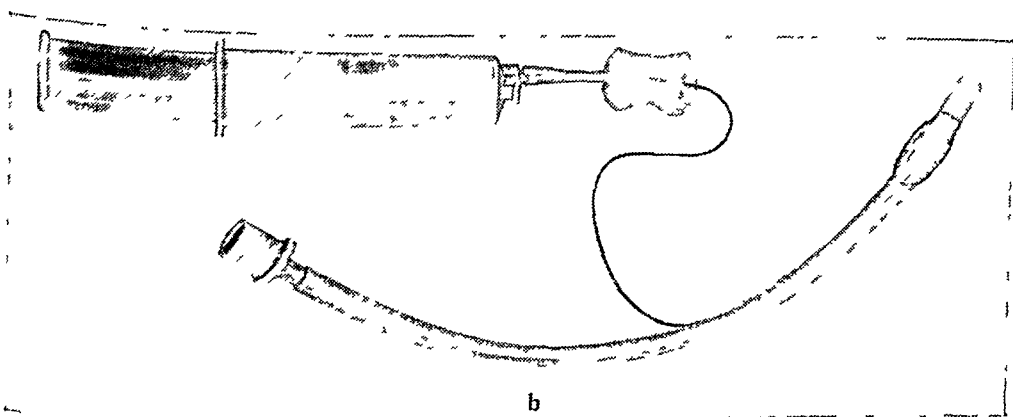
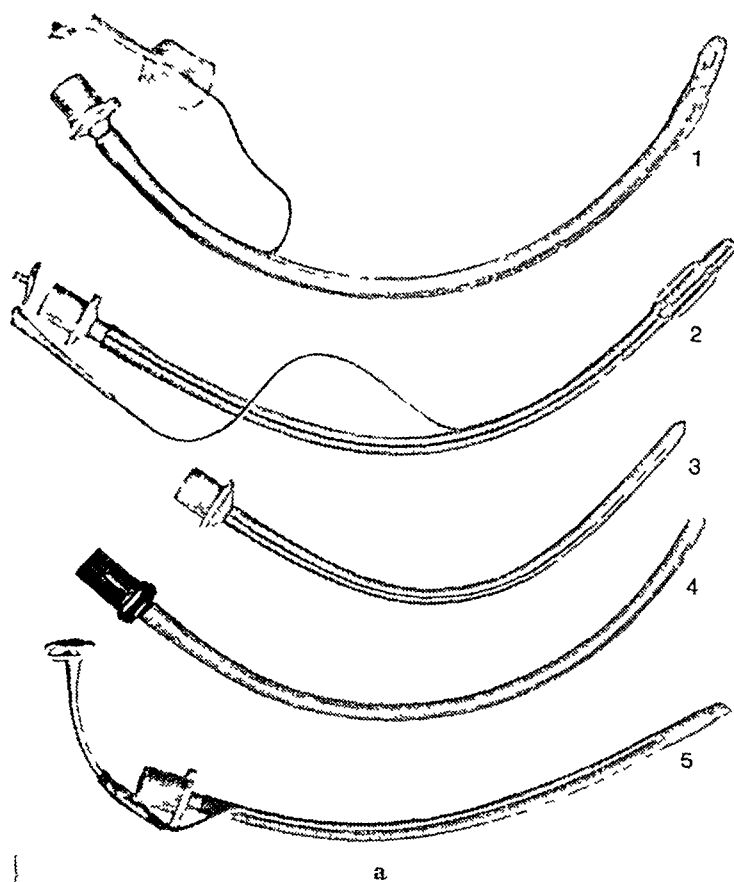


Fig 7 Endotracheal tubes for children

a—(1) plastic tube with an inflatable cuff for right upper lobe bronchus, (2) plastic tube with an inflatable cuff, (3, 4) plain plastic tubes, (5) reinforced tube, b—inflating a cuff

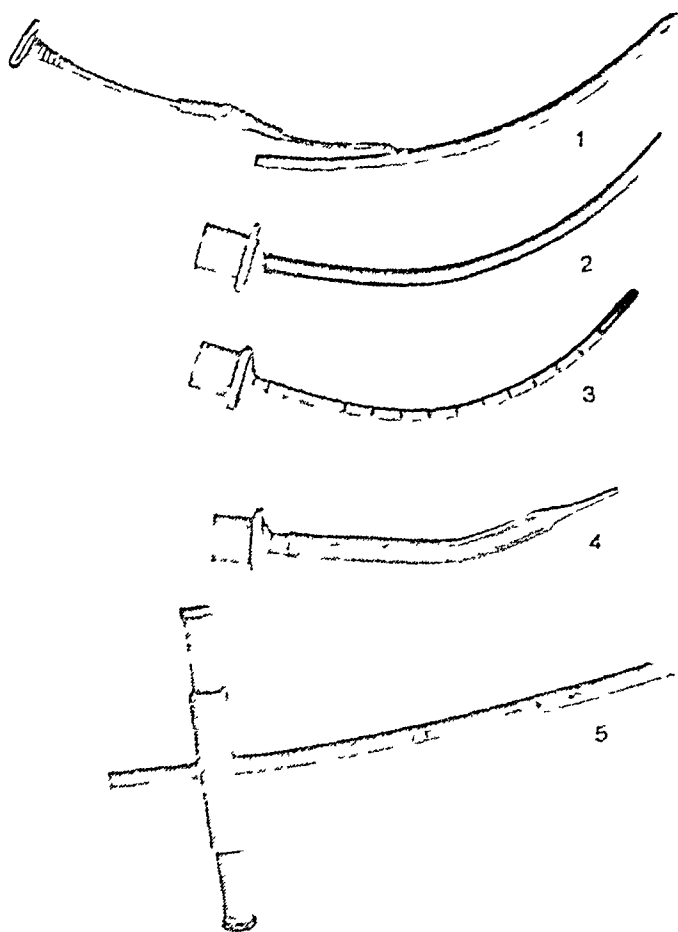


Fig 8 Endotracheal tubes suitable for use with neonates

1—tube for upper right bronchus, with an inflatable cuff, 2 and 3, plain tubes, 4—Cole's tube, 5—tube for nasotracheal intubation

*Face masks* of various size are now available and it is possible to select a mask that would tightly fit the patient's face to cover only the mouth and the nose, thus decreasing the dead space

*Airways* are special devices used for inhalation anaesthesia with a face mask, they are intended to prevent occlusion of the vocal slit with the tongue root

*Endotracheal tubes* (Figs 7 and 8) are made of special rubber or plastics, with and without inflatable cuffs, with one or two lumens. Sizes of the endotracheal tubes are given in Table 1

*Tracheostomy tubes* are made of metal, rubber, or plastics. They are manufactured with and without cuffs (Fig 9)

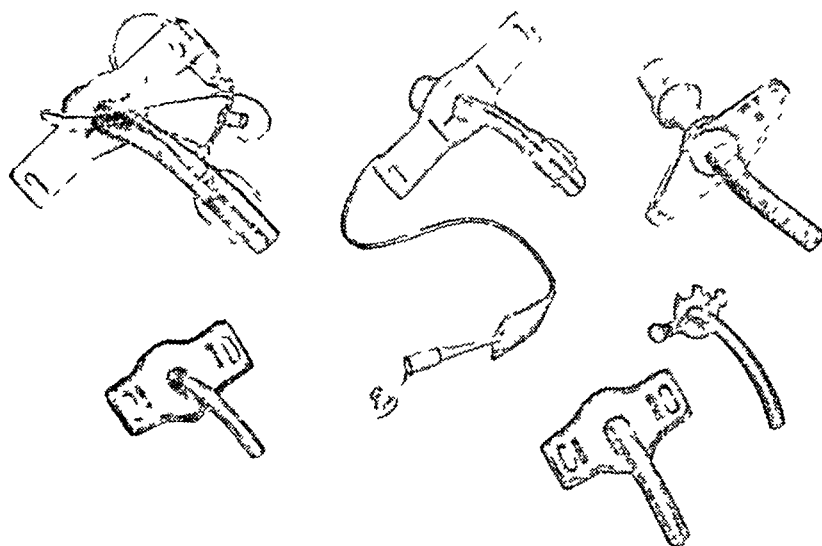


Fig 9 Tracheostomy tubes

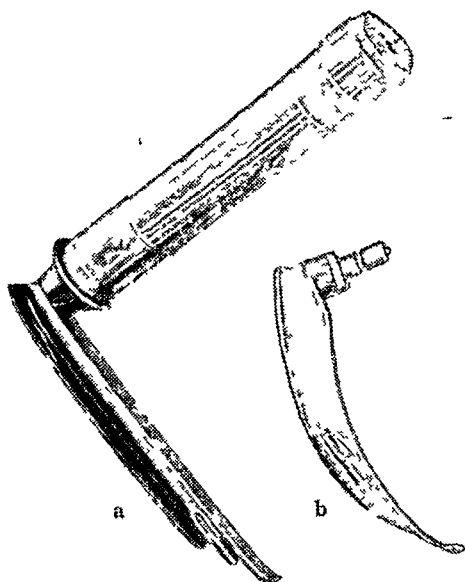


Fig 10 Laryngoscope for older children  
(a) with straight and (b) curved blade

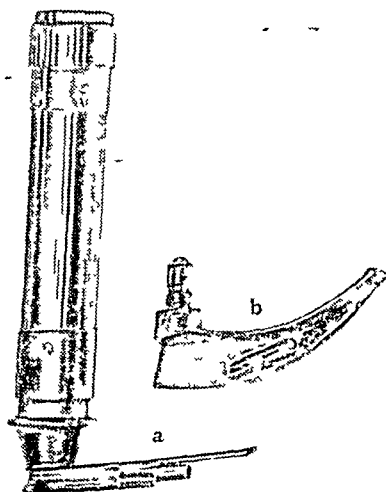


Fig 11 Laryngoscope for infants  
with (a) straight and (b) curved blade



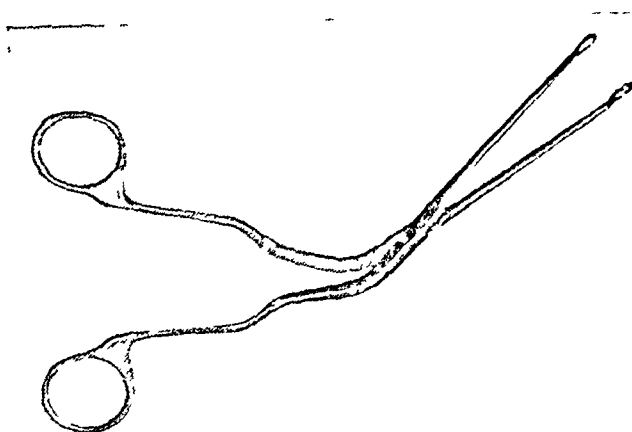


Fig 12 Magill forceps

*Laryngoscope* is an apparatus used for inspection of the larynx and for tracheal intubation. The instrument includes a handle with enclosed electric cells, a blade, and an electric lamp. Straight and curved blades of various sizes (Figs 10 and 11) are available. Direct laryngoscopy of children is conducted with small blades, or using

Table 1 Sizes of Endotracheal Tubes Depending on the Child Age

| Age     | Outer dia ,<br>mm | Tube length, cm        |                         | Marking |        |            |
|---------|-------------------|------------------------|-------------------------|---------|--------|------------|
|         |                   | for oral<br>intubation | for nasal<br>intubation | Soviet  | Magill | Charrriere |
| Neonate | 4 3-5 0           | 10-11                  | 12-12 5                 | 00      | 00     | 13-15      |
| 6m      | 5 3-5 6           | 10 5-11 5              | 13                      | 0       | OA-0   | 16-17      |
| 1y      | 6 0-6 3           | 11-12                  | 13-14                   | 1       | 1      | 18-19      |
| 2y      | 6 6-7 0           | 12 5-13 5              | 14-15                   | 2       | 2      | 20-21      |
| 3y      | 7 3-7 6           | 13-14 5                | 15-16                   | 3       | 3      | 22-23      |
| 5y      | 8 0-8 3           | 14-16                  | 18-19                   | 4       | 4      | 24-25      |
| 9y      | 9 3-9 6           | 16-17 5                | 20-21                   | 6       | 6      | 28-29      |

a special paediatric laryngoscope. A skilled anaesthesiologist has no problems with using any blade, either straight or curved, but a newcomer to laryngoscopy should better use a straight blade.

A *Magill forceps* (Fig 12) facilitates intubation of the trachea. It is used to guide the end of the endotracheal tube through the vocal slit. The Magill forceps is usually used for intubation through the nose.

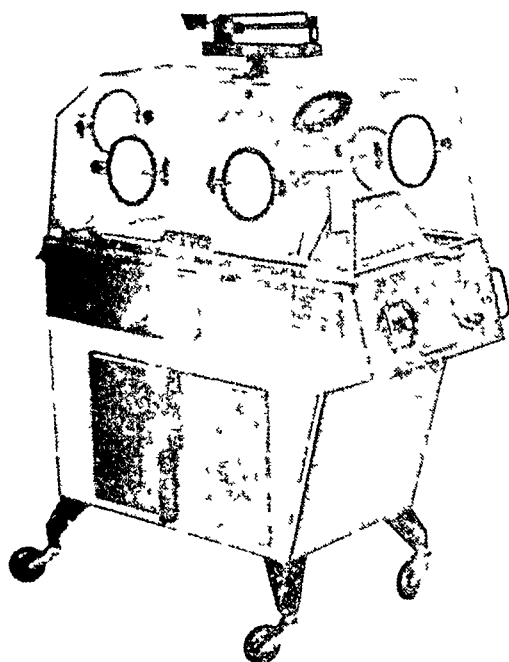


Fig 13 Couveuse for neonates ('Medikor Hungary)

Couveuses (incubators) are used to provide optimum climatic conditions. They are intended for premature neonates or infants who need intensive therapy. The needed oxygen concentration, air humidity, and temperature are maintained in such couveuses. A couveuse encloses a cot for an infant, which is covered with a transparent cover to separate the child from the environment. Medical personnel can conduct minor procedures through special windows.

Table 2 Dependence of Air Temperature in the Couveuse on Body Weight During the First 24 Hours of Infant's Life

| Body weight, g | Mean temperature, °C |
|----------------|----------------------|
| 500            | 35.5 ± 0.5           |
| 1000           | 34.9 ± 0.5           |
| 1500           | 34.9 ± 0.5           |
| 2000           | 33.5 ± 0.5           |
| 2500           | 33.2 ± 0.8           |
| 3000           | 33.0 ± 1.0           |
| 3500           | 32.8 ± 1.2           |
| 4000           | 32.6 ± 1.4           |

in the cover Table 2 specifies temperatures that are recommended for the initial stage of infant care, the relative humidity of the atmosphere in the couveuse being 50 per cent. Lower air humidity requires higher ambient temperature.

Three types of couveuses are available. Couveuses of the first type are intended for incubation of premature neonates or newborns with small body weight (Fig 13). These are usually used in maternity houses. Microclimate is maintained in such couveuses and the neonate can also be given oxygen or aerosol therapy. Couveuses of the other type are intended for neonates in critical condition or post-operative patients. The couveuses are provided with additional devices and attachments such as a vacuum sucker, temperature sensors, inhaler, etc. Various manipulations, such as X-ray examination, intubation of the trachea, artificial ventilation of the lungs, and minor operations can be conducted on an infant inside the couveuse.

The third type of a couveuse is intended to maintain the necessary conditions inside the cot during transportation of the child in a critical condition. During transportation the infant can be given oxygen or aerosol therapy, infusion therapy, or artificial lung ventilation, his liquid secretions can be removed by

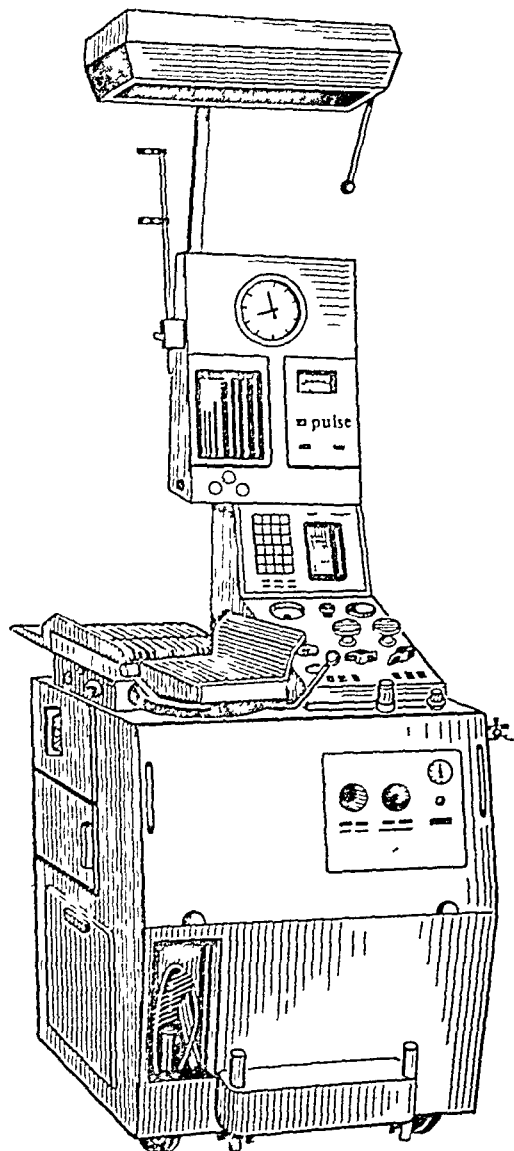


Fig 14 Castor-mounted resuscitation apparatus ('PARK')

suction, the respiratory and circulatory functions of the baby can be constantly maintained. The chamber where the baby is placed is easy to open. Since the cover is transparent and easily removable, the child can be given immediate aid in case of emergency.

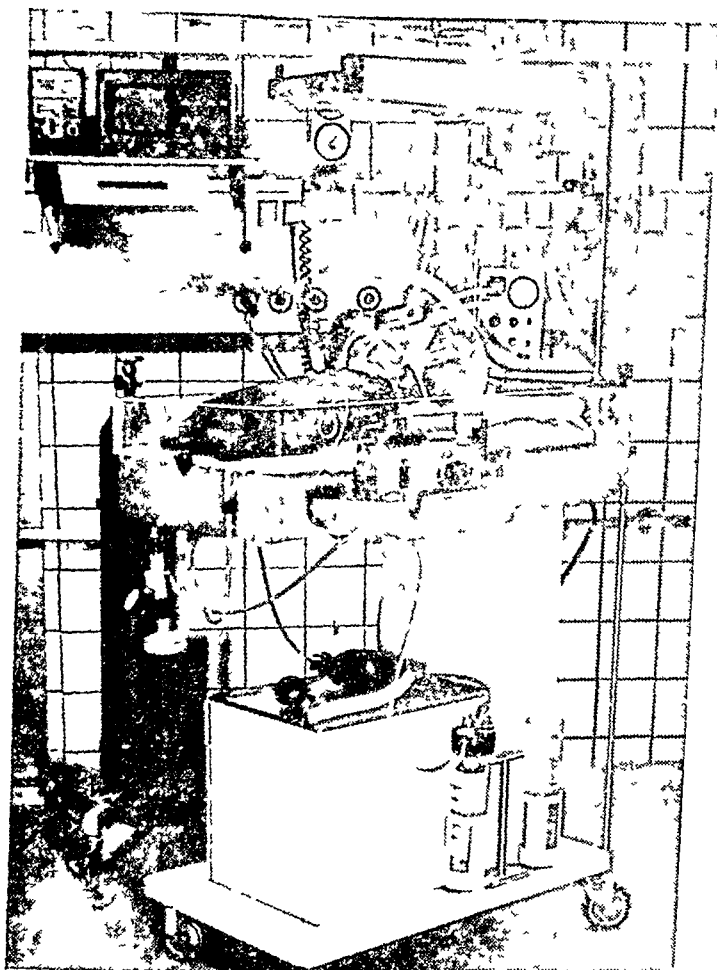


Fig 15 Resuscitation unit for neonates

A special device for accumulation of energy can maintain normal conditions inside the chamber for at least 2 hours in case of accidental interruption of energy supply from the mains

Special resuscitation units should be available for intensive therapy of neonates. The unit should be provided with a heater to provide normal temperature, devices for suction of liquids, apparatus for artificial ventilation of the lungs, a monitoring unit, and other devices that may be needed during puncturing, venesection, cannulation of veins, laryngo- and bronchoscopy, dressing, etc. Castor-mounted resuscitation unit 'PARK' (Soviet-made) is used in this country (Fig 14) The same principle operates in foreign-made resuscitation units (Fig 15)

## MONITORING

The vital functions of children in critical condition should be under constant observation. This can be ensured with monitoring devices which follow changes in the ECG, arterial pressure, circulation of blood, respiration, body temperature, and concentration of  $\text{CO}_2$  and  $\text{O}_2$  in the inhaled and exhaled air (Fig. 16). Special monitors are used for transcutaneous determination of oxygen and carbon dioxide tension (Fig. 17).

*Special devices are used for accurate dosing of medicinal fluids for infusion.* These are drop counters, syringe and peristaltic pumps. The capacity of these devices can be accurately adjusted in the range from 0.5 to 299 ml/hr (for a 50-ml syringe). The high accuracy can be maintained for long-term infusions. The devices ensure sterility of the infusion system. As the syringe is emptied, an automatic device switches off the system and emits a light and sound signal. Peristaltic pumps can be used with children of all ages. Their capacity can be adjusted to any rating within the range from 1 to 999 ml/hr.

**Inhalation therapy** can be given to children of any age. The apparatus used for inhalation of aerosols includes a mixer, a transparent hood or box, and a humidifier. Inhalation therapy includes also inhalation of electric aerosols of aqueous medicinal solutions and

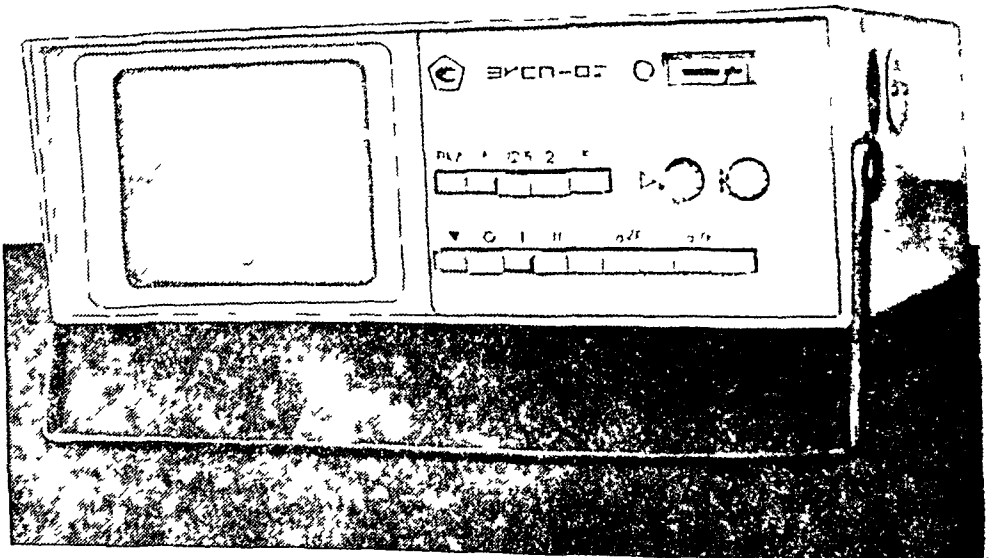


Fig. 16 Portable electrocardioscope

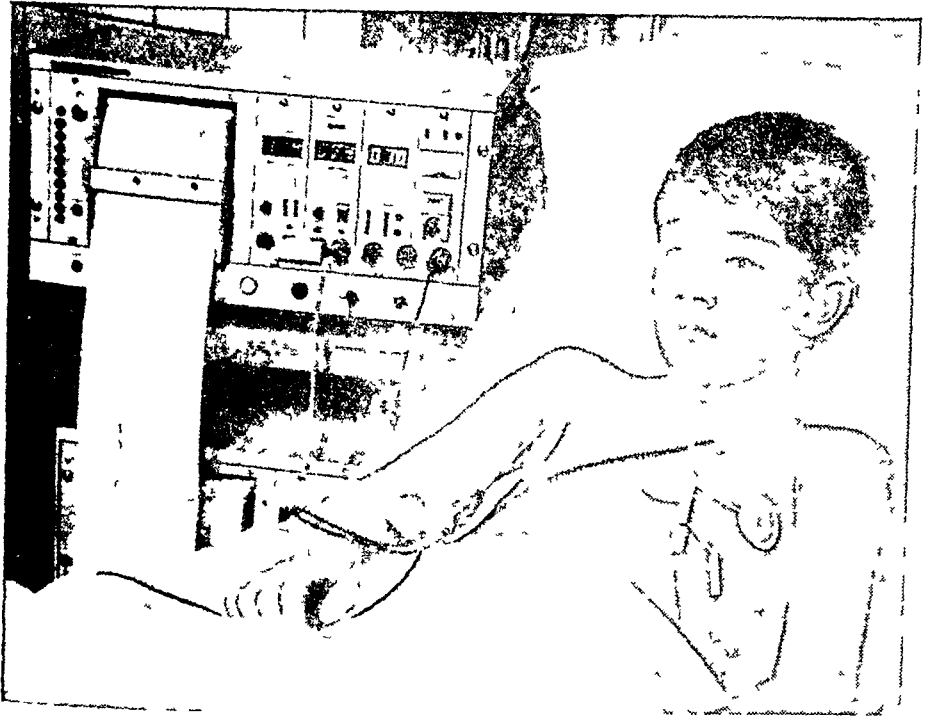


Fig 17 Transcutaneous monitor for determining oxygen and carbon dioxide tension in the blood

oils Inhalers can be used with children of any age and in any condition and are safe for both the patient and the attending personnel. Inhalers are available in which liquid media, e.g. water, water-soluble phytoncides, antibiotics, enzymes, spasmolytics, etc., are atomized by ultrasound ensuring greater therapeutic effect, because particles sizing 1-5  $\mu\text{m}$  penetrate the minutest bronchi.

### HYPERBARIC OXYGENATION

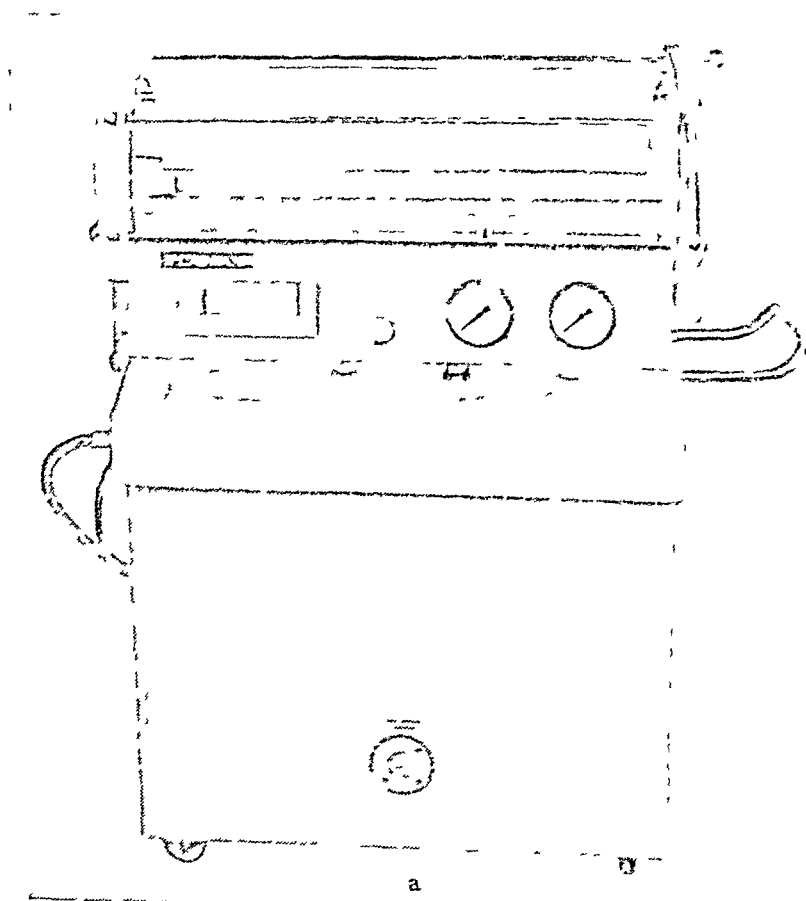
Children and adults can be treated with oxygen under excess pressure. Chambers for infants under 1 year of age have the capacity to 70 litres, and oxygen consumption in them varies between 30 and 35 l/min, the pressure that can be attained in these chambers is to 3 atm. The required humidity and constant temperature are maintained in the chamber. A provision is made for emergency decompression and elimination of  $\text{CO}_2$  (Fig 18).

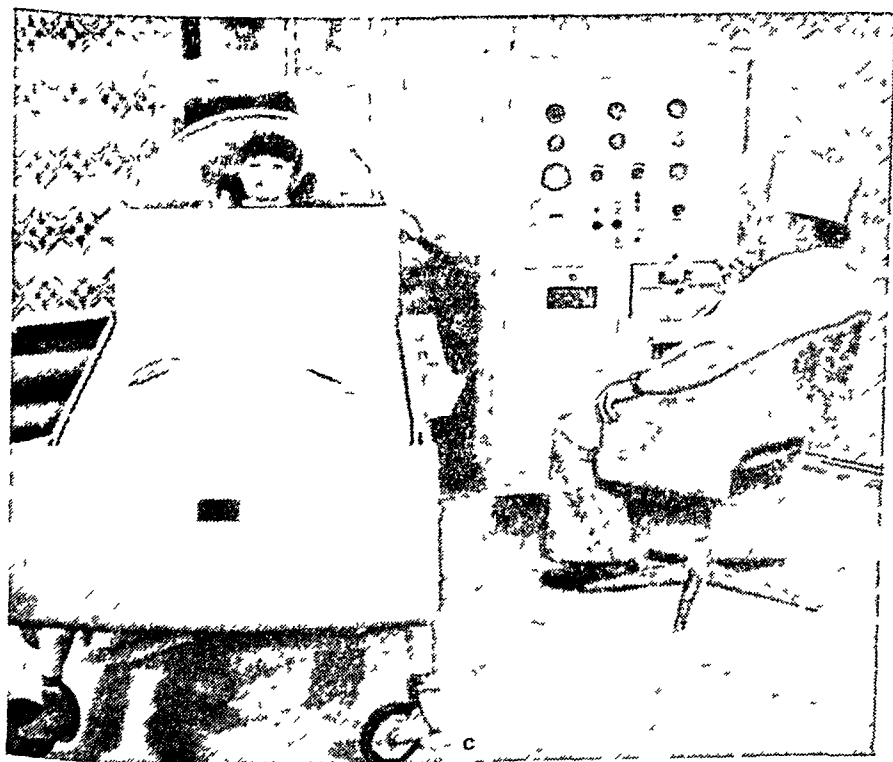
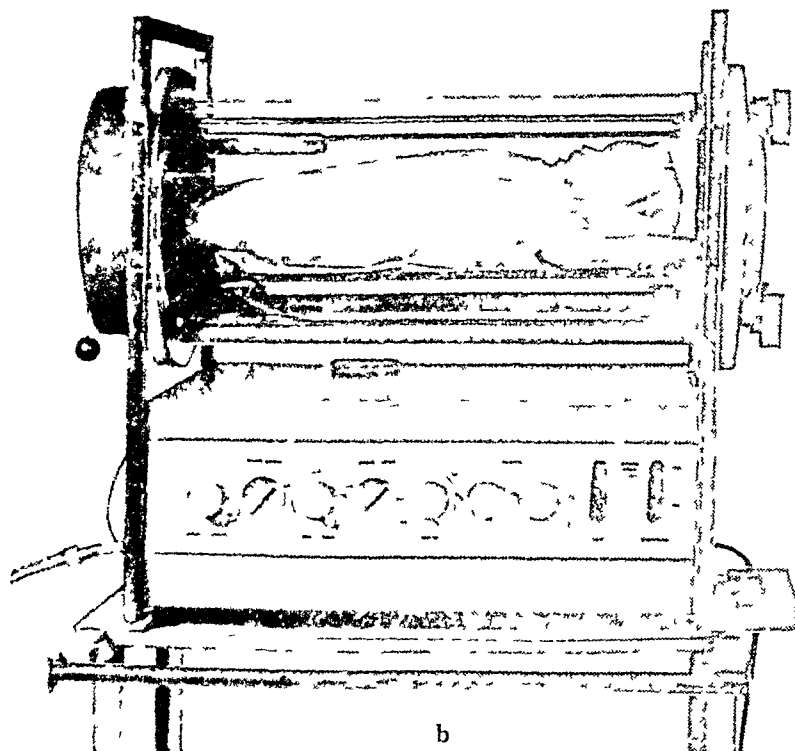
## EQUIPMENT OF AMBULANCE CAR FOR TRANSPORTATION OF CHILDREN IN CRITICAL CONDITION

Transportation of children in critical condition is a very complicated problem. Special temperature conditions should be provided inside the ambulance car (Fig. 19). If necessary, the child's lungs should be artificially ventilated and various manipulations performed. Short-term surgical operations included. It follows, therefore, that the ambulance car should be equipped at least with the following:

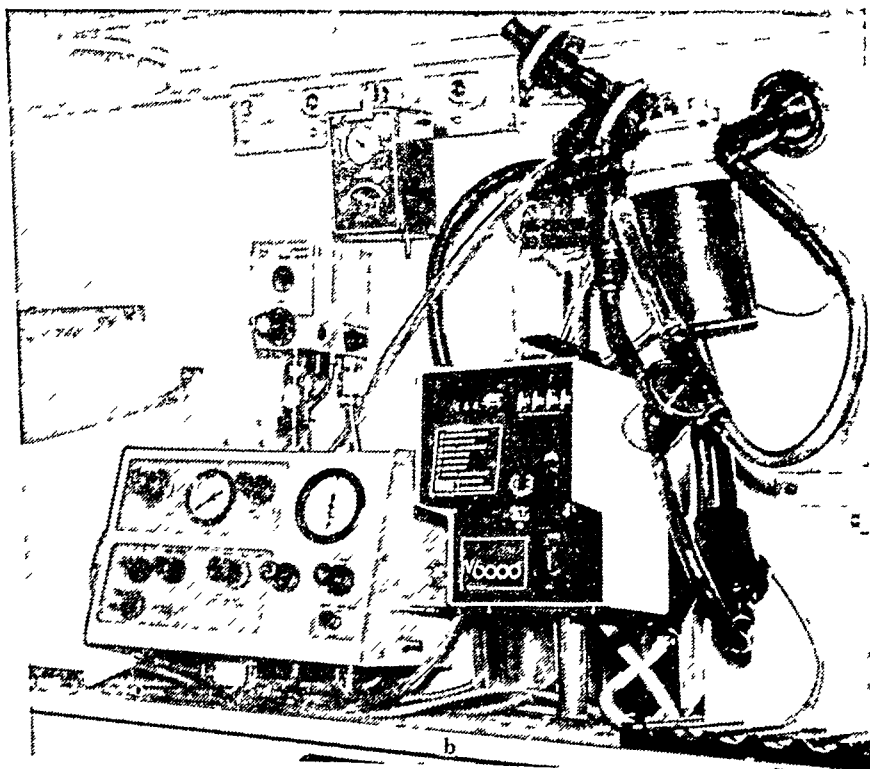
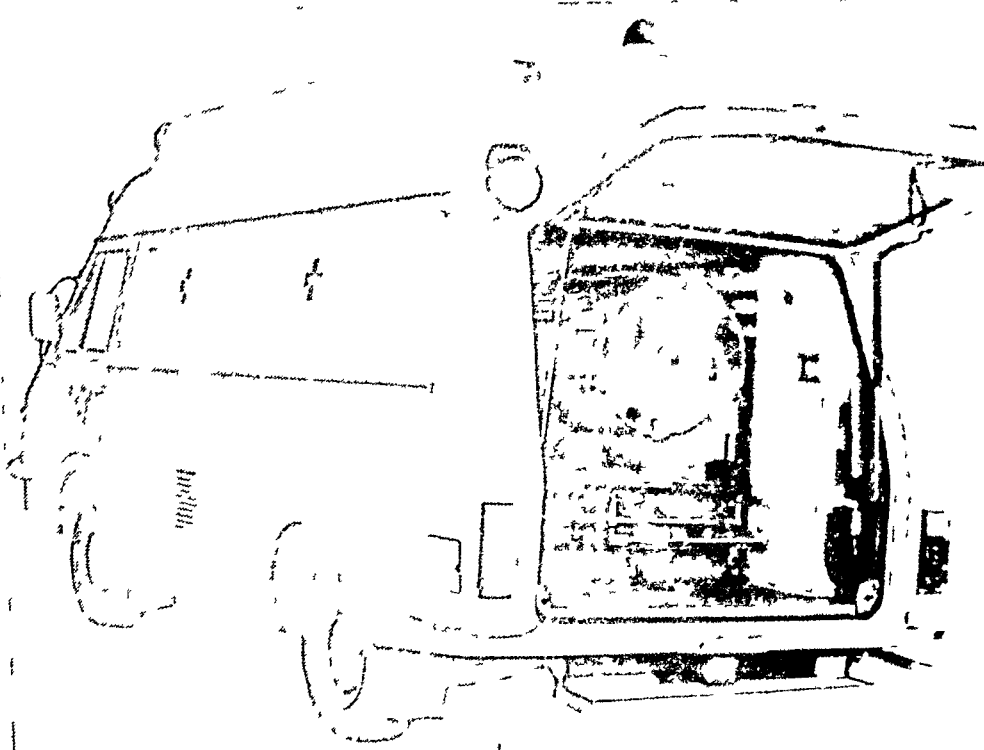
- 1 A couveuse
- 2 Oxygen cylinders with reducing valves and a mixer where oxygen is mixed with air
- 3 A compressed air cylinder

Fig. 18 Chambers for hyperbaric oxygenation of infants (a), neonates (b) and older children (c)









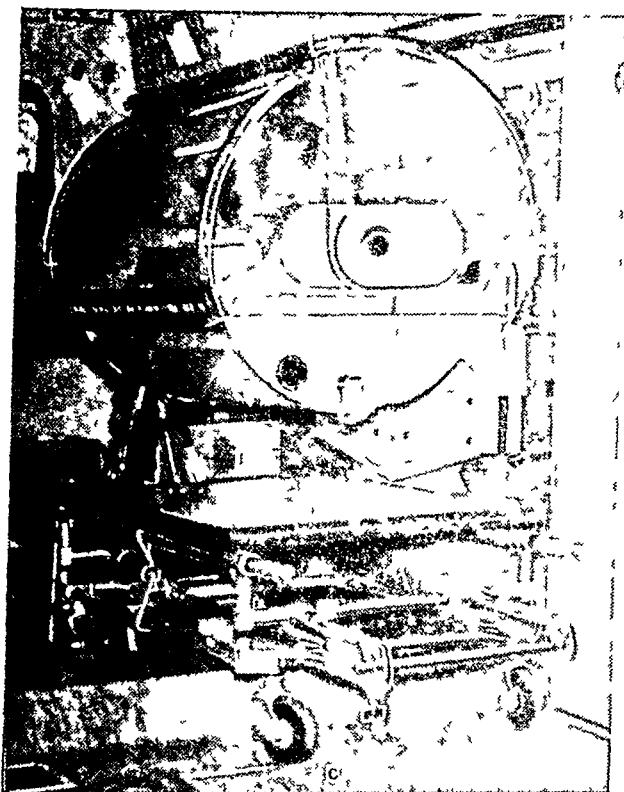


Fig 19 Ambulance car for transportation of neonates

(a) general view, (b and c) inside the car

- 4 A monitor to control the action of the heart (with skin electrodes)
- 5 A monitor to control body temperature (with skin and rectal sensors)
- 6 An apparatus for measuring arterial pressure.
- 7 A vacuum pump
- 8 An air bag with valves and face masks
- 9 An apparatus for artificial lung ventilation with intermittent positive pressure ventilation and continuous positive airway pressure
- 10 A set of endotracheal tubes with adapters and connectors.
- 11 A laryngoscope with sets of straight and curved blades
- 12 A system for intravenous infusions, complete with a shaver and needles for puncturing veins of the head
- 13 A pump with a complete set of systems for intravenous infusions
- 14 A defibrillator
15. Sterile sets of tools for section of veins and arteries, for cerebrospinal and pleural puncturing, tracheotomy

- 16 An apparatus for aspiration of fluids from the pleural cavity
- 17 Sterile gloves, surgical tools and instruments, suturing silk, cloths, syringes, and gastric tubes
- 18 Medicines

### Chapter 3

## Anatomy and Physiology of a Child as Related to Anaesthesiology and Intensive Therapy

Anatomy and physiology of a child are described in detail in special textbooks, and in this chapter we shall therefore only discuss those aspects of children's anatomy and physiology that are especially important for anaesthesiology and intensive therapy of children.

### NERVOUS SYSTEM

The anatomical and structural immaturity of the nervous system of a child accounts for certain functional features of the body that should be remembered by an anaesthesiologist. The insufficient differentiation of the nerves and the small number of the interneuronal connections explain the inadequate regulating effect of the cortex on the dependent parts of the central nervous system. Most reflexes are therefore realized through the subcortical formations. This explains the fact that neonates react by reflex and stereotype to various stimuli, including pain, without differentiation of their character or strength. The response to a comparatively weak stimulus is often diffuse, strong and generalized. This mainly explains the tendency of infants to develop convulsions.

Predisposition of an infant to convulsive responses is also explained by insufficient myelinization of the nervous fibres, increased water content of the cerebral tissue, and more intensive metabolic processes. An anaesthesiologist should remember that permeability of the blood-brain barrier and the hydrophilic properties of the brain are high in infants. Even a moderate hyperhydration can cause swelling and oedema of the brain with various subsequent complications. The functional immaturity of the central nervous system is also manifested by the activity of the vegetative nervous system. The main manifestations of this immaturity are high lability and inadequate control of the vital functions, such as respiration, muscular activity, or thermal regulation. The absence of the functional reserve and the rapid 'fatigue' of the nervous system of infants are mostly manifested by various respiratory disorders.

A paediatric anaesthesiologist should be well aware of the special character of the infant's psychic properties. Persistence of a 2-3 year-

old child or negativism of an adolescent can become a serious obstacle to the preparatory anaesthetic procedure or other manipulations. Psychotherapy and even sedatives and ataractics are often necessary.

The spinal cord of a neonate extends to the 3rd lumbar vertebra. Only by the end of the first year it reaches the 1st lumbar vertebra, which should be remembered when conducting diagnostic lumbar puncture.

### RESPIRATORY SYSTEM

**Anatomy.** The nasal cavity of a child is narrower than in an adult, while the bottom of the cavity is so inclined that the tongue contacts the posterior wall of the pharynx over a larger surface. The nasal mucosa is thin, richly invested with vessels, and has no developed cavernous tissue. This accounts for rare nasal bleeding in neonates.

The larynx of a neonate is comparatively high, at the level of the 3rd cervical vertebra. The anatomical relationships between the tongue, the epiglottis, and the larynx are an obstacle to direct laryngoscopy or intubation, and present difficulties in using a curved Macintosh blade.

The narrowest point in the airways is the trachea at the level of the cricoid cartilage. The mucosa thickens at this point by 1 mm, e.g. in catarrhal inflammation, to decrease the lumen by 75 per cent in neonates against 20 per cent in older children. This explains the special danger of oedema of the mucosa in infants, which can cause a complete obstruction of the airways.

The right bronchus is thicker and shorter and is continuous with the trachea. If a tube is passed deeper than required, it usually gets into the right main bronchus. The angle between the bronchus and the trachea is usually the same in children and adults.

The ratio of the anatomical dead space to the tidal volume is about the same (0.3) in humans of all ages. But the absolute volumes in infants are low and this provides conditions for a dangerous increase in the share of the dead space during anaesthesia or artificial lung ventilation.

The lungs of a neonate are rich in connective tissue, they are plethoric and less elastic than in an adult. A neonate born at term has about 24 million alveoli and their number is tripled by the third month. (An adult has 296 million alveoli.) The diameter of an alveolus in a neonate is 5-6 times smaller than in an adult. The overall gas-exchange surface in an adult is 20 times as great as in a neonate, which agrees with the ratio of weights of an adult and a neonate. It is easy to understand that even a small amount of liquid secretion in the alveoli impairs the respiratory function.

**Lung capacity.** In order to characterize the respiratory function, the total lung capacity (the volume of air contained in the lungs at

the end of a maximal inspiration) is divided into some static volumes and capacities. The volume of air which is inspired and expired in normal quiet breathing is called the tidal volume. The volume of complementary air that can be inspired after a normal inspiration is called the inspiratory reserve volume. The expiratory reserve volume is the supplemental air that can be expelled from the lungs after a normal expiration. The volume of air that remains in the lungs after a maximal expiratory effort is called the residual volume. The volume of air that can be expired from the lungs after a maximal inspiration is called the vital lung capacity. The residual lung capacity plus the expiratory reserve capacity is the functional residual capacity. The tidal volume plus the inspiratory reserve volume is the inspiratory capacity. The functional residual capacity, the vital lung capacity, and the total lung capacity characterize mainly the mechanical properties of the lungs. Maintenance of the optimum gas composition of the alveolar air is mainly connected with the ventilation volume and the functional residual capacity of the lungs because the gas exchange is intensified with enlargement of the functional residual lung capacity.

In order to distend the lungs during the first inspiration of a neonate, the air pressure of 6-8 cm  $H_2O$  should be created inside the lungs. The pressure drops to zero at the end of expiration but part of air remains in the lungs creating the functional residual volume. All subsequent inspirations do not require this high pressure because the lungs now remain partially distended at rest.

The functional residual capacity of the lungs depends on the interaction of two forces one of which tends to distend the lungs (expansion of the chest) and the other force accounts for the tendency of the lungs to collapse. A state of equilibrium is established after a normal expiration the pressure in the pleural cavity is about  $-5$  cm  $H_2O$  in older children and in adults, in neonates this pressure is  $-1$  or  $-2$  cm  $H_2O$ .

Narcosis, surgical intervention, distension of the abdomen or diseases of the lung can change the pulmonary volumes. Since the organs of the abdominal cavity can change their position, the functional residual capacity of the lungs is smaller when a person is in the prone or dorsal position (than in sitting or standing position). The functional residual capacity decreases also in patients with some diseases of the lungs associated with respiratory disorders due to decreasing elasticity of the pulmonary tissue. Quite the reverse, the functional residual capacity increases in diseases characterized by spasms of the airways associated with formation of 'air traps', as is the case with bronchial asthma. Moreover, the functional residual capacity is a buffer that diminishes the variations in oxygen and carbon dioxide tension in the blood during the respiratory cycle, while the air remaining in the lungs after expiration prevents,

in a certain measure, the collapse of the alveoli. Transpulmonary pressure of 4-5 cm  $H_2O$  is sufficient to maintain normal tidal volume, while the pressure required to expand collapsed lungs, e.g. after thoracotomy, is about 15-25 cm  $H_2O$ .

**Lung ventilation.** Pulmonary ventilation is a volume of gas delivered into the airways per unit time. A normal inspiration is an active process realized by contraction of the diaphragm, intercostal and (during exercise) accessory muscles. A normal expiration occurs due to a spontaneous contraction of the lungs, the chest, and relaxation of the diaphragm. Expiration of neonates, even at rest or during sleep, is an active rather than passive process in which flexor muscles of the spine, the intercostal and the abdominal muscles are involved.

The comparatively high respiratory rate of a neonate (34 per minute) can be explained by the tendency of the body to equilibrate the respiratory rate and the tidal volume so that the oxygen demand might be met with the minimum energy consumption.

Enlargement of the tidal volume during exercise or in pathological conditions is limited in a neonate due to the horizontal arrangement of the ribs: the chest is constantly in the position of inspiration and the diaphragm is high. The minute ventilation of the lungs in infants therefore increases mainly due to accelerated respiratory rate. The work of breathing in an adult increases by 50 per cent with doubled respiration rate, while a healthy neonate can increase the respiratory rate to 60 per minute without substantial change in the work of breathing. But a further increase in the respiration rate markedly increases the energy consumption and soon causes decompensation of the respiratory function.

Only part of air inspired into the lungs is involved in gas exchange because only part of the inhaled air enters the perfused alveoli. The alveolar ventilation is therefore the vital characteristic of gas exchange occurring in the lungs. The remaining part of the total gas exchange is dead space. The total (physiological) dead space is usually classified as anatomical and alveolar dead space.

Anatomical dead space is the volume of airways. Normally it is  $\frac{1}{3}$  tidal volume and is well correlated with the anthropometric findings. The volume of the anatomical dead space can change significantly in various pathological conditions depending on the degree of airway obstruction and inflammation in the lungs. The anaesthesiologist should remember that some pharmacological preparations (atropine, aminophylline) and anaesthetics can also change the dead space.

Since the absolute volumes of dead space are small in infants, the anaesthesia apparatus and respirators should be selected very carefully: even insignificant enlargement of dead space will significantly diminish the alveolar ventilation.

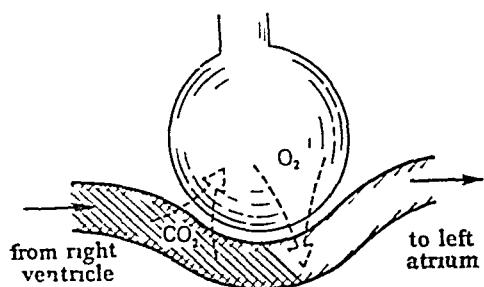


Fig 20 Gas diffusion through the alveolar wall

The difference between functional and anatomical dead space is alveolar dead space. The alveolar dead space is very small in a healthy individual and the physiological dead space is therefore practically equal to the anatomical dead space. The alveolar dead space is enlarged during ventilation of non-perfused alveoli, e.g. in embolism of the pulmonary artery, or in cases of marked prevalence of ventilation over perfusion. The most common causes of enlargement of dead space are haemorrhages and hypotension: part of the ventilated alveoli are not perfused due to redistribution of the pulmonary circulation. Another cause of enlargement of dead space is increased mean pressure in the airways, e.g. in artificial lung ventilation or spontaneous respiration with positive end expiratory pressure and the presence of sites with very high ventilation-perfusion ratio in the lungs.

**Gas diffusion in the lungs** Passage of gases through the alveolar-capillary membrane obeys the laws of diffusion (Fig 20). The amount of gas passed through the lung membrane per unit time, i.e. the diffusion rate, is directly proportional to the difference of partial pressure of the gases separated by the membrane, and inversely proportional to the diffusion resistance. Resistance of gas diffusion in the lungs is characterized by its reciprocal conduction, which is called diffusion capacity of the lungs. This is the amount of gas passing through the alveolar-capillary membrane per minute, the difference between the partial pressure of gases separated by the membrane being 1 mm Hg. The diffusion capacity is proportional to the lung capacity. Consequently, not the absolute figures characterize the diffusion capacity in paediatric practice but their relation to the functional residual capacity; this characteristic is about the same in children of all ages.

Disturbed gas diffusion between air and blood is usually important for the oxygen exchange since the solubility of oxygen and its diffusion capacity is 20 times lower than that of carbon dioxide. Furthermore, diffusion is considered completed when oxygen combines with haemoglobin. It has been established that this stage is about 50 per cent of the total diffusion resistance.

Disturbances in the diffusion capacity of the lungs caused by

impaired permeability of the alveolar-capillary membrane occur in patients with oedema of the lungs, in hyaline membrane disease, interstitial pneumonia, and some other diseases. But more pronounced disorders in the diffusion capacity occur with decreasing surface area of effective gas exchange, e.g. in conditions following vast resection of the pulmonary tissue, and more frequently in severe disturbances of the ventilation-perfusion relationship.

**Ventilation-perfusion relationship.** The efficiency of pulmonary gas exchange depends not only on the absolute values of the alveolar ventilation or pulmonary blood flow but mostly on the relationship between these two values. During the first day of life perfusion of the lungs prevails over ventilation. Later, the overall ventilation-perfusion ratio becomes the same as in adults, 0.8. Three variants of ventilation and blood circulation can be shown schematically as follows:

1. The alveolar ventilation and blood flow are even ( $V_A/Q = 0.8$ ). In this case the blood flowing from the alveoli has the normal gas composition.

2. Ventilation prevails over blood flow ( $V_A/Q > 0.8$ ). This occurs during hyperventilation of the perfused alveoli or during normal ventilation with decreased blood flow. The blood flowing from the alveoli contains normal amount of oxygen and decreased amount of carbon dioxide.

3. Alveolar ventilation is less than blood flow ( $V_A/Q < 0.8$ ). This situation is possible with decreased ventilation and unchanged blood flow, or if the blood flow increases over normal values. Arterial hypoxaemia develops under these conditions. Partial pressure of carbon dioxide in the arterial blood usually remains normal due to its high diffusion capacity.

During an operation and anaesthesia the overall and regional ventilation-perfusion relationships change. An important factor causing these changes is a prolonged motionless position of the patient during operation. The vital lung capacity of a lying patient decreases by about 8-10 per cent due to the high position of the diaphragm.

Artificial lung ventilation, inhalation of hyperoxic breathing mixtures, anaesthetics, muscle relaxants, and some other medicines are also very important for the ventilation/perfusion ratio. It should be noted that artificial lung ventilation and the position of the patient during operation are essential mostly for the regional distribution of ventilation, and anaesthetics have their effect mostly on the regional perfusion.

Pulmonary perfusion can be disturbed following massive blood transfusion. Pathogenesis of these disorders is connected with obstruction of the pulmonary capillaries by aggregations of the formed elements of the blood (erythrocytes, leucocytes, thrombo-



cytes) These are always contained in the 'old' blood but they can also be formed inside the vessels, especially in hypotension and shock These disorders occur in gas and fat embolism

Distribution of pulmonary blood flow can undergo substantial changes in decreased pressure in the pulmonary artery Alveolar perfusion in the upper parts of the lungs is discontinued and the alveolar and physiological dead space increase Pressure in the pulmonary artery can decrease due to administration of ganglioblocking preparations and due to decreased volume of circulating blood (in profuse bleeding or loss of liquid)

The utmost disturbance of the ventilation-perfusion ratio is intrapulmonary arteriovenous shunting This occurs in complete discontinuation of alveolar ventilation with unchanged blood flow Ventilation discontinues and atelectases develop mostly during bronchial occlusion, early expiratory closure of the airways, or primary collapse of the alveoli associated with decreased surfactant level Whichever may come first—the alveolar collapse or the expiratory closure of the airways, atelectasis occurs finally, because the gas trapped in the alveolus diffuses into the blood in all cases The rate at which the alveolus collapses depends on the character of the gas filling it. This rate is several times higher during breathing pure oxygen or helium-oxygen mixtures than during air breathing

**Surface tension and surfactant** The stability of the pulmonary alveoli is maintained by a surfactant, which is synthesized by mitochondria of the alveolar epithelium The surfactant is a complicated lipoprotein, which equilibrates the forces of surface tension, thus preventing collapse of the alveoli As the diameter of the alveoli decreases during expiration, the layer of the surfactant thickens and it thus resists more actively the surface tension forces When the alveoli expand, the surfactant layer thins and its activity lessens Neonates are very sensitive to the surfactant system insufficiency The diameter of the alveoli in a neonate is several times smaller than in an adult and, according to the Laplace law, the surface tension increases with decreasing volume of a bubble, and the easier the bubble collapses

Surfactant deficiency may occur in various pathological conditions, such as hypoxia, upset microcirculation in the lesser circulation, marked reduction in the cardiac output, etc The surfactant is inactivated by hyperoxic breathing mixtures, aspiration of the stomach contents, and some toxic substances

**Mechanics of breathing** The gas exchange between the alveolar air and the environment is normally effected by rhythmical contractions of the respiratory muscles The muscular effort determines the volume and rate of gas movement, the relationships between these indices can, therefore, be described using the laws of mechanics Movement of air along the airways meets the resistance of

two types the elastic and non-elastic (aerodynamic) resistance.

The term 'compliance' is used in clinical physiology to characterize the elastic properties of the lungs and the chest, which is the reciprocal of elasticity. Compliance ( $C$ ) is determined as the change of volume per unit of pressure change  $C = \frac{\Delta V}{\Delta P}$  and is expressed in litres per 1 cm H<sub>2</sub>O. Compliance changes significantly with age; compliance of a newborn is 0.004 l/cm H<sub>2</sub>O and of an adult, 0.15 l/cm H<sub>2</sub>O.

Compliance depends on the morphological properties of the lungs and the chest, the volume of blood in the pulmonary vessels, the tone of the thoracic and abdominal muscles, the amount of the lung tissue involved in the gas exchange, and the bronchial tone. Compliance decreases with decreasing airiness of the pulmonary tissue and formation of micro-atelectases. Changes in compliance are even more pronounced in hyaline membrane disease, lung oedema, atelectases, and the shock lung syndrome.

Non-elastic, or aerodynamic, resistance is determined by the pressure difference which is required to ensure a given air flow rate

$$R = \frac{\Delta P}{V} \text{ cm H}_2\text{O}/(\text{l/s})$$

Aerodynamic resistance depends on the length ( $l$ ) and radius ( $r$ ) of airways and also viscosity of gas ( $\eta$ ), the relationship between these factors is expressed by the Poiseuille formula

$$R = \frac{8l\eta}{\pi r^2}$$

It is evident that the radius of the airways is the most important factor for the aerodynamic resistance, and that resistance, therefore, depends on age too. The main cause of increased resistance are conditions characterized by spasms or obstruction of the airways. These conditions are croup, bronchitis, bronchial asthma, and the like.

The aerodynamic resistance can increase significantly during anaesthesia if artificial airways are used (endotracheal tube, artificial respiration apparatus). All these factors increase the work of breathing.

**Other functions of the lung.** Apart from the gas-exchange function, the lungs perform some other very important functions as well. The lungs are, first of all, a filter that purifies the blood from pathological matter such as cell aggregations, fibrin clots, etc. Owing to the presence of enzyme systems, these impurities are retained in the lungs and metabolized.

The lungs also perform an important role in the coagulating and anticoagulating systems of the blood. Heparin and thromboplastin are produced in them. In addition to heparin, the mast cells of the alveoli produce some biochemically active substances, mainly

histamine, which is involved in regulation of perfusion of the lungs and other organs. Vasoactive kinins, whose content dramatically increases in shock and septic conditions, are inactivated in the lungs. Epinephrine passes through the lung filter, while norepinephrine is retained and destroyed in the lungs. Hypothermia and deep anaesthesia can decrease the norepinephrine-inactivating power of the lungs, thus causing spasm of peripheral vessels and microcirculatory disorders. Being an elastic reservoir, the lungs are a very important organ regulating the circulating blood volume and maintaining continuity of blood flow.

### CARDIOVASCULAR SYSTEM

The pulmonary blood circulation in a foetus is very small, it is 10 per cent of the right ventricle output. The blood saturated with oxygen is delivered from the placenta through the umbilical vein and the venous ducts into the inferior vena cava where it combines with a small portion of blood outflowing from the lower extremities and the abdominal organs. The blood further enters the right atrium, passes through the foramen ovale into the left atrium and then through the left ventricle into the aorta. The greater portion of the pulmonary blood flow bypasses the lungs (from the right to the left) through the ductus arteriosus since the pulmonary vascular resistance is much higher than the systemic. As the first breath is taken and the umbilicus is compressed, the pressure in the right atrium falls. The pulmonary blood circulation thereby increases and the pressure in the left atrium increases too, as a result of which the foramen ovale is closed functionally. Increased partial tension of oxygen in the arterial blood, the fall in the  $\text{CO}_2$  tension, and the rise in the pH decrease most effectively the pulmonary resistance.

The functional closure of the ductus arteriosus in a healthy neonate occurs in 10-15 hours following birth. But the anatomical closure occurs in 2-3 weeks, after which normal 'adult'-type circulation is established.

Adverse effects, such as hypoxaemia, hypercapnia, acidosis or release of catecholamines constrict the vessels and increase vascular resistance in the lungs. Non-closed ductus arteriosus and the foramen ovale can begin functioning again. In the presence of severe hypoxaemia, to 80 per cent of the cardiac output can be bypassed through the anatomical or pulmonary shunts (50 per cent through the ductus arteriosus). This condition is common for hyaline membrane disease or congenital diaphragmatic hernia.

Breathing mixtures containing high concentration of oxygen, prostaglandins, and  $\beta$ -adrenergic blocking agents, such as tolazoline, decrease hypertension in the lesser circulation and promote functional closure of the ductus arteriosus and the foramen ovale.

The heart rate of a neonate is quite stable. Sinus arrhythmia and

high variation in the pulse rate are quite frequent in infants but serious arrhythmias are rare

The arterial pressure in infants is much lower than in adults. A healthy neonate has a systolic pressure between 65 and 70 mm Hg, it increases by 10-15 mm Hg during the first two weeks of life. Although it is difficult to measure arterial pressure in neonates, an anaesthetist must remember that infants tend to develop shock due to hypovolaemic hypotension.

The heart rate of children is higher than in adults and may vary within broad range, causing no complications. The pulse of a resting neonate is 90-110 per minute but it quickly increases to 170-180 when an infant cries. Atropine premedication increases the heart rate to 170-190 per minute. Immediately after birth the cardiac output is 400-500 ml/kg per minute, it then decreases in the early postnatal period. The output of the left ventricle is first higher than of the right ventricle because part of blood is shunted through the open oval foramen and the arterial canal. When the shunts are closed, the outputs of both ventricles become equal (150-200 ml/kg per minute).

The volume of circulating blood in a neonate is relatively higher than in an adult. It is 10 per cent of the body weight during the first months of life (80-110 ml/kg), while in infants under 1 year of age it is 8.5 per cent, and in children of preschool age, 7-8 per cent. But since the total volume of blood in a neonate does not exceed 350-400 ml, a loss of 50 ml of blood corresponds to a loss of 1000 ml in an adult.

The capillary system of infants is characterized by insufficient development of muscular elements in arterioles and precapillary sphincters. Arteriovenous anastomoses are, therefore, the main regulators of the peripheral circulation. In stress conditions, the blood circulation in neonates is usually centralized, while microcirculation and tissue blood supply are disturbed.

Oxygenation of blood of a neonate is higher than of an adult because the haemoglobin content of a neonate blood is higher. The foetal haemoglobin prevails in neonates. Its concentration during the first day of life is about 70 per cent, and by the end of the second week it decreases to 50 per cent. The foetal haemoglobin has higher affinity for oxygen; it combines with oxygen more readily and gives it off with greater difficulty. Moreover, foetal haemoglobin is readily converted into methaemoglobin.

### URINARY SYSTEM

Table 3 gives mean volumes of urine excreted at a time by persons of various ages. The absolute amounts of excreted urine during 24 hours increase with age, while the relative values decrease. Table 4 shows absolute and some relative values of daily urinary excretion in infants and children.

Table 3 Volumes of Urine (single micturition)  
Depending on Age

| Age     | Excreted urine, ml |
|---------|--------------------|
| to 6 m  | 30                 |
| 1 y     | 60                 |
| 3-5 y   | 90                 |
| 7-8 y   | 150                |
| 10-12 y | 250                |

Table 4 Daily Absolute and Relative Volumes of Excreted Urine

| Age     | Absolute volume, ml | per kg body weight, ml | per sq m of body surface, ml |
|---------|---------------------|------------------------|------------------------------|
| 1-3 d   | 0-96                | 0-30                   | to 480                       |
| 4-5 d   | 1-217               | —                      | —                            |
| 6-12 d  | 1-355               | 54                     | 810                          |
| 1-6 m   | 170-670             | 39-98                  | 694-1836                     |
| 7-12 m  | 175-810             | 21-81                  | 565-1840                     |
| 1-5 y   | 600-900             | 45-60                  | 1169-1364                    |
| 6-10 y  | 700-1200            | 35-39                  | 909-1091                     |
| 10-14 y | 1000-1500           | 30-32                  | 909-1000                     |

It has been established that the kidneys of neonates and infants under 1 year of age are immature anatomically and functionally. Some renal functions become comparable with those of adult kidneys (or equal to them) only at the age from 6 months to one year. In some respects the renal function becomes adequate only by the age of two years. Table 5 characterizes the renal function of children of various ages.

Table 5 Functional Characteristics of the Kidneys

| Age                | Rate of glomerular filtration, ml/min $\times 1.73 \text{ m}^2$ | Maximum concentrating power in dehydration, mOsm/l | pH      |
|--------------------|---|--|---------|
| Premature Neonates | 2-40  | 190-680  |         |
| 1-2 d              | 8-42  | 180-650  | 4.9-6.8 |
| 4-12 d             | 20-60   | —  | 5.5-7.4 |
| 15-30 d            | 30-90   | 200-1100   | 5.3-6.6 |
| 2 m-1 y            | 42-160  | 400-1330   | 4.9-7.3 |
| 1 y-5 y            | 100-235   | 400-1510   | 5.3-6.7 |
| 6-12 y             | 88-170  | 400-1430   | 5.7-6.8 |

Chronic diseases of the kidneys occur less frequently in children than in adults. Acute renal failure is also less frequent in children and it converts less frequently into chronic renal failure. The prognosis of infectious allergic diseases of the kidneys is better in children than in adults. Glomerulonephritis of streptococcal aetiology ends in complete recovery in about 95 per cent of the cases.

### GASTROINTESTINAL TRACT

The tongue of a neonate is short, thick and broad, which may interfere with intubation of the trachea or during laryngoscopy. By the end of the first year of life these features of the tongue disappear which is probably connected with discontinuation of breast-feeding. Appreciable salivation is established by the 4-6th month of life, which commonly coincides with the time when an infant is introduced to mixed feeding.

Anaesthesia, intensive therapy and resuscitation manipulations may be connected with intubation, and the anaesthesiologist therefore must know the structure of the oesophagus. The dimensions of the oesophagus are therefore important (Table 6). The distance from the teeth to the cardia can be calculated using the following formula:  $1/5 \times \text{body length (cm)} + 6.3 \text{ (cm)} = \text{the length of the oesophagus (cm)}$

Table 6 Dimensions of the Oesophagus Depending on Age

| Age      | Length, cm | Diameter, cm |
|----------|------------|--------------|
| Neonates | 10         | 0.7-0.8      |
| 1 y      | 12         | 1.0          |
| 5 y      | 16         | 1.2-1.5      |
| 10 y     | 18         |              |
| 15 y     | 19         |              |

The cardinal sphincter of a neonate is weak physiologically, while the muscular layer of the pylorus is powerful. This accounts for regurgitation and vomiting, and should be remembered when conducting general anaesthesia and administering muscle relaxants to neonates: the regurgitated material can be aspirated by the neonate and cause severe complications, such as aspiration pneumonia. Lethal outcomes are possible. Regurgitation can also be due to specific configuration of the stomach. Although the stomach shapes are not constant, the stomach position is usually horizontal.

The capacity of the stomach increases with age till the child grows 1 or 2 years old. Later growth of the stomach depends not

Table 7 Capacity of the Stomach  
in Infants

| Age      | Capacity, ml |
|----------|--------------|
| Neonates | 30-35        |
| 3 m      | 100          |
| 1 y      | 250          |

only on age but also on the eating habits. The capacity of the stomach is given in Table 7.

The values are only tentative, especially for pathological conditions. For example, in obstruction of the upper portions of the intestine, the capacity of the stomach can increase 2-5 times.

The motor function of the stomach in normal conditions depends on the character of nutrition and also on the neuroreflex impulses. The high activity of the vagus and the splanchnic nerves stimulates spasm of the stomach and the pylorus, respectively.

### WATER-ELECTROLYTE METABOLISM

Body weight, surface area and energy metabolism units are the main criteria for assessing the condition of the water electrolyte metabolism. The body-weight unit is the most convenient for practical assessment of the metabolism, especially in infants to 5 years of age.

Absolute water content of the infant body increases with age (to comply, to a certain degree, with increasing anthropometric indices).

The relative content of water is higher in infants and it decreases with age. This decrease is especially marked during the first six months of life, later the decrease becomes less pronounced. As an infant grows to the age of 2 or 3 years, his relative water content becomes the same as in an adult. Table 8 characterizes the water content with respect to the body weight of children.

Table 8 Water Content of a Child's Body

| Age    | % of body weight | Age     | % of body weight |
|--------|------------------|---------|------------------|
| 0-1 d  | 79.0             | 1-2 y   | 58.7             |
| 1-10 d | 74.0             | 2-3 y   | 63.5             |
| 1-3 m  | 79.3             | 3-5 y   | 62.2             |
| 3-6 m  | 70.1             | 5-10 y  | 61.5             |
| 6-12 m | 60.4             | 10-16 y | 58.0             |

For convenience of assessment of the water-electrolyte metabolism, water (fluid) spaces are distinguished in the human body. The main are the extracellular and intracellular spaces. The extracellular space also includes the intravascular space, in which water is the main component. It is a solvent for the ions and proteins and also the medium for dispersion of suspended cells of blood.

The absolute volumes of blood, plasma, and erythrocytes increase with the growth of the infant, while the total amount of blood and plasma changes insignificantly with respect to the overall weight of the body. The amount of blood relative to the body weight varies in children (except neonates during their first day of life) between 7.3 and 9.1 per cent. The total amount of blood may be 11-13 per cent of the body weight during the first day of life. The amount of plasma is even smaller: 4.6 to 5.1 per cent of the body weight.

**Water exchange between the body and the environment.** The equilibrium between the water uptake and withdrawal is decisive for the stability of the water exchange between an infant and the environment. The amounts of liquid taken and discharged are different in infants, which is normal and is explained by the growth of the body. This difference accounts for 65-70 per cent of the daily weight gain of an infant. This phenomenon is especially pronounced during the first six months of life, during the period of most intense growth of the infant's body. Water is uptaken in two forms: as free water of food (for example, breast milk contains about 87 per cent water) and as bound water, i.e. water which is formed during oxidation of nutrients. This water is also called endogenic, or water of combustion (metabolic water). It has been established that excretion of water from the body is proportional to the amount of energy released by the body. Infants lose 45 ml of water through perspiration and exhalation per each 100 kcal of released energy, this figure is 20-22 ml in 16-year-old children, water lost from the body with the urine is 50-75 ml and with faeces, 5-10 ml. The amount of water lost with perspiration is from 0 to 25 ml (depending on the temperature of the body and the ambient temperature) per 100 kcal of released energy. The water of combustion (metabolic water) is 12 ml. The mean demand for water is thus about 100 ml per 100 kcal.

The relative water demand with respect to the body weight unit differs significantly in children depending on their age. Table 9 gives data on mean water demands depending on age. Variations of water demand with the size of the body surface area are less pronounced.

**The ionic composition of body fluids.** The fluids of the extra- and intracellular spaces of the body vary significantly in their composition and concentration of the ions. Sodium is the main cation of the extracellular fluid, while potassium of the intracellular fluid. The concentration of the chloride ion is the highest in the extra-



Table 9 Water Demands Depending on the Age of a Child

| Age     | Water demand,<br>ml/kg×day | Age  | Water demand,<br>ml/kg×day |
|---------|----------------------------|------|----------------------------|
| 1 d     | 20-30                      | 9 m  | 125-145                    |
| 2 d     | 30-40                      | 1 y  | 120-135                    |
| 3 d     | 40-60                      | 2 y  | 115-125                    |
| 4 d     | 60-80                      | 4 y  | 100-110                    |
| 5 d     | 90-110                     | 6 y  | 90-100                     |
| 6 d     | 115-125                    | 10 y | 70-85                      |
| 7 d     | 140                        | 14 y | 50-60                      |
| 7 d-3 m | 140-160                    | 18 y | 40-50                      |
| 6 m     | 130-155                    |      |                            |

cellular fluid Magnesium stands the second, after potassium, in the list of cations contained in the intracellular fluid, while its content in the extracellular fluid is very low The main anion of the extracellular fluid is the chloride ion, while organic phosphates and proteins are the main anions of the intracellular fluid. Table 10 gives concentrations of the main ions in the extra- and intracellular fluids of the body.

Table 10 Concentration of the Main Ions in the Intracellular, Extracellular and Interstitial Fluids (mmole/litre)

| Ion                        | Body Fluid    |               |              |
|----------------------------|---------------|---------------|--------------|
|                            | Extracellular | Intracellular | Interstitial |
| Sodium                     | 135-145       | 10-25         | 135-145      |
| Potassium                  | 3.8-5.5       | 130-150       | 4.5-5.5      |
| Magnesium                  | 0.5-1.5       | 15-20         | 0.5-1.0      |
| Calcium                    | 2-2.5         | —             | 1.5-2.0      |
| Chloride                   | 105-110       | —             | 110-120      |
| Bicarbonate                | 20-25         | 8-12          | 25-30        |
| Protein                    | 16            | 65            | —            |
| Organic acids              | 6             | 6             | —            |
| Sulphate                   | 0.5           | 12.5          | 0.5          |
| Phosphate, monosubstituted | 1.0           | 50.0          | 1.0          |

It should be noted that the concentration of ions does not practically vary with the age of children, and significant deviations from the tabulated data are only possible in neonates during their adaptation to the extrauterine life These transient deviations are associated with various factors such as the mother's condition before parturition, character of pregnancy and labour, the general reaction of

the neonate to the change of the environment, the mode of feeding, water uptake during adaptation, etc

**Main ion demands** The demands for the main ions per unit weight of the body decrease slightly with increasing age. The normal sodium demand of a neonate is 3-5 mmole/(kg/day) and it decreases gradually to 2-3 mmole/(kg/day) in 5-10 year-old children, while this demand falls to 1 mmole/(kg/day) in 13-14 year-old children, which is about the same as in adults.

The daily potassium demand is 2-3 mmole/kg for a neonate, 2 mmole/kg for 6-12 months to 8-12 year-old children, 1.5 mmole/kg for children during their prepubertal and pubertal periods, and 1 mmole/kg for adults. The chloride ion demands are met practically simultaneously with potassium and sodium demands.

**Osmosis and osmotic pressure.** The selective passage of solvent (water, in the human body) through a semipermeable membrane is called osmosis. Osmosis depends on osmotic pressure, which is proportional to the concentration of the solute, the absolute temperature, the permeability of the membrane to a given solute, and the effective concentration of this solute in solution. Osmotic pressure of a solution, whose solute molecule falls into several particles (electrolytes) depends on the number of particles into which the solute molecule falls during dissolution.

The main osmotically active substances of the extracellular fluid are sodium, chlorine, the bicarbonate ion, and anions of organic acids. Molecules of some other substances, e.g. glucose and urea, are also important. Their concentration can increase significantly in some pathological conditions, e.g. diabetes mellitus, renal failure. Osmotic pressure of intracellular fluid is determined by concentration of potassium, magnesium, phosphate, and small uncharged molecules.

Osmotic pressure created by very large molecules, e.g. of protein, man-made polymers, is called oncotic pressure. Normally the oncotic pressure depends on the presence of proteins. It is small and averages 0.03-0.04 atm. Under normal conditions, the vascular wall is impermeable to proteins, and therefore the interstitial fluid has practically no oncotic pressure. The difference in protein concentration gives an insignificant gradient in osmotic pressure between the plasma and the interstitial fluid, but is very important for the water exchange between these fluids.

Common instruments fail to detect differences in osmotic pressure of fluids in the body cavities. The pressures of the extra- and intracellular fluids are, therefore, considered to be equal.

Osmotic pressures of solutions are compared (without determining them) by their osmolar concentration.

**Osmolarity of body fluids** Osmolarity, or osmolar concentration, is the concentration of osmotically active particles in solution. The unit of osmolarity is osmol.  $\text{Osmol} = C \text{ mole} \times n$ , where  $C$  is the

amount of solute, and  $n$  is the number of particles into which a molecule of the solute dissociates. For non-dissociating substances  $n = 1$ . A thousandth fraction of osmol is commonly used in practice. A milliosmol (mOsm) =  $C \text{ mmole} \times n$ .

The osmolar concentration corresponds to the number of milliosmols per unit mass or volume of solution. Osmolarity can be used for rough calculations of osmotic pressure of various solutions and for comparison of osmotic pressure of solutions and fluids. Osmolar concentration of body fluids varies within a narrow range. For the main fluid spaces it normally varies between 285 and 310 mOsm/litre. The mean osmolarity of neonates and infants (to 1 year of age) varies within these limits but their maximum values may vary within a wider range. The variation range of a 2-year-old infant falls within the specified limits.

### ACID-BASE BALANCE

The acid-base balance of the internal medium of a body is an important characteristic of homeostasis. It is determined by the ratio of concentrations of some substances involved in maintenance and regulation of the acid-base balance, and also by the function of the excretory organs, especially of the lungs and the kidneys. The main characteristic of the acid-base balance is the reaction of the liquid media of the body characterized by the hydrogen ion concentration. The active reaction of the body fluids is normally maintained within narrow limits in children of all ages. A normal neonate excretes and neutralizes at least 30 litres of hydrogen. It should be noted in this connection that the main function of the acid-base system is neutralization of hydrogen liberated during oxidation processes.

The activity of the hydrogen ion in the plasma in a normal body is  $40 \pm 5$  mequiv/litre. For convenience, the hydrogen ion concentration is expressed by the negative logarithm of its reciprocal (pH). Normal pH is between 7.35 and 7.45. The maximum values of pH, at which life is feasible and which can be reversed by appropriate treatment of children, are 6.8 and 7.8.

**Buffer systems** A buffer couple, or simply a buffer, is a solution containing a weak acid and the conjugated base, which is formed in solution due to dissociation of this acid. The presence of a buffer prevents marked variations in the pH of solution when an acid or alkali are added. The best buffer properties are observed with the concentration of acid corresponding to the logarithm of the reciprocal of the dissociation constant of this acid. The pK of carbonic acid in the human body is 6.10. The reaction of the medium is determined by the Henderson-Hasselbalch equation

$$\text{pH} = \text{pK} + \log \frac{[\text{HCO}_3^-]}{[\text{H}_2\text{CO}_3]}$$

Under normal conditions the concentration of  $\text{H}_2\text{CO}_3$  is 1.2 and of the  $\text{HCO}_3^-$  ion, 24 mmole/l. Then

$$\text{pH} = \text{pK} + \log \frac{2}{1.2} = 6.10 + \log 20 = 7.4$$

The ratio of concentrations of carbonic acid and the bicarbonate (mainly sodium bicarbonate  $\text{NaHCO}_3$ ) in the extracellular fluid determines its pH.

The bicarbonate system  $\frac{\text{H}_2\text{CO}_3}{\text{NaHCO}_3}$  is the main buffer. It is 53 per cent of the overall buffering power of the blood. But the human body has other buffers: the phosphate buffer  $\frac{\text{NaH}_2\text{PO}_4}{\text{Na}_2\text{HPO}_4}$  (its buffering power is low even if the organic phosphate is also considered) and the albumin buffer in which both the plasma albumins  $\frac{\text{H albumin}}{\text{B albumin}}$  and albumins of erythrocytes  $\frac{\text{H haemoglobin}}{\text{B haemoglobin}}$  (where B stands for a univalent cation, e.g. sodium or potassium cation) are involved.

The active reaction (pH) of the body can be altered only by substances in dissociated rather than in their molecular state. Acids dissociating almost completely ( $\text{HCl} \rightleftharpoons \text{H}^+ + \text{Cl}^-$ ) and giving many free hydrogen ions are strong acids and can shift the pH to the acid side. Weakly dissociating acids ( $\text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^-$ ) are in their molecular state in the body and cannot cause considerable changes in the pH. Likewise, there are strong and weak bases.

All buffer systems are mixtures of weak acids and alkaline salts of these acids. Their dissociation is insignificant and when in the human body, they do not alter the pH, and the body can therefore hold them in store.

The mechanism of action of all buffer systems consists in conversion of all strong acids or bases, which may be uptaken by the body from outside or generated inside the body, into weak acids and weak bases.

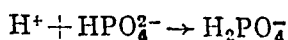
|      |   |  |   |
|------|---|--|---|
| HCl  | $\text{NaHCO}_3$<br>alkaline<br>buffer    | NaCl<br>neutral<br>salt                        | $\text{H}_2\text{CO}_3$<br>weak<br>acid |
| NaOH | $\text{H}_2\text{CO}_3$<br>acid<br>buffer | $\text{NaHCO}_3$<br>weakly<br>alkaline<br>salt | $\text{H}_2\text{O}$<br>water           |

The active reaction (pH) of the blood does not practically change in either of these cases.

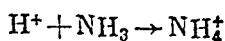
In addition to the buffer (physico-chemical) systems, the physiological systems, such as the lungs, kidneys, or the gastrointestinal tract, are also involved in maintenance and regulation of activity of liquid media in the body. Carbon dioxide is withdrawn from the body by pulmonary ventilation. The respiratory centre controls the

ventilatory function of the lungs so that carbon dioxide tension ( $\text{PCO}_2$ ) in body fluids is maintained at 40 mm Hg. The concentration of the bicarbonate ion  $\text{HCO}_3^-$  is controlled by the kidneys. The tubular reabsorption of anions is controlled so that the anion deficit of 24-27 mmol/l is constantly maintained. The deficit is compensated by the bicarbonate formed by dissolution of metabolic  $\text{CO}_2$  in body fluids in quantity corresponding to its partial pressure and body temperature.

The kidneys regulate the acid-base balance in two ways by forming bicarbonate from carbon dioxide and water with involvement of carboanhydrase, and by withdrawal of the hydrogen ion. Hydrogen is excreted by the kidneys in the bound form, such as conjugated acids of buffers, e.g. those formed according to the equation



or in the form of ammonium, which is produced by the reaction between ammonia and hydrogen



The first reaction shows the formation of the titrated acidity of the urine. The ammonium ion has no effect on the reaction of the urine, i.e. the second reaction ensures withdrawal of the hydrogen ion without a further reduction of the urine pH. Under normal conditions hydrogen is withdrawn in exchange for sodium reabsorption, and these processes are, probably, conjugated. Anions of nonvolatile acids are withdrawn from the body by simple filtration in the glomeruli, the anions are practically not reabsorbed.

**Acid-base balance in infants.** The tendency to acidosis (accumulation of the hydrogen ion) is the special property of an infant body. This is connected with a relatively intense production of hydrogen in metabolic processes. On the other hand, the factors responsible for the acid-base balance in a healthy infant are similar to those of an adult. The only exception are neonates during the period of adaptation to the extrauterine life. The hydrogen ion concentration in the body fluids of infants is high during this period, the pH and the compensatory pressure of  $\text{CO}_2$  in the body fluids decrease accordingly. It should be noted that irrespective of age, the function of all systems responsible for the acid-base control ensures the conditions in which a healthy infant may live.

**Determining the acid-base balance of an infant.** This is commonly determined not by directly measuring the hydrogen ion concentration in the body fluids, but by determining some integral values of concentration of substances important for the acid-base balance and by using special nomograms. Practically the acid-base balance depends on the acid-base balance of extracellular fluid as determined by pH and  $\text{PCO}_2$  of plasma by two methods. According to one of them, pH

and  $\text{PCO}_2$  of blood are determined simultaneously using special electrodes, with subsequent calculation of the sought values by nomograms. Modern apparatuses can do without nomograms they give either direct readings on the screen or have them typed on paper.

**Characteristics of acid-base balance.** Following below are complete characteristics of the acid-base balance of a human body that are used to assess changes in the acid-base balance. pH is the integral index characterizing practically the active reaction of the extracellular fluid,  $\text{PCO}_2$  is the carbon dioxide tension in the blood (in the plasma or the extracellular fluid, which actually is the same). This shows if the lung function corresponds to the rate of formation of carbon dioxide and protons in the body or (tentatively) the ventilation capacity of the lungs, BE is the shift of the buffer bases, it shows the excess or deficit of the buffer bases (conjugated acid anions) and characterizes metabolic regulation of the acid-base balance; BB characterizes the overall concentration of the buffer bases of the blood, SB is the value characterizing concentration of the standard bicarbonate of plasma, i.e. in standard conditions of temperature and pressure ( $37^\circ\text{C}$  and  $\text{PCO}_2 = 40$  mm Hg), AB is the actual bicarbonate, the value showing the concentration of the plasma bicarbonate at a given temperature and carbon dioxide pressure at the moment of blood taking. The normal indices are shown in Table 11.

Table 11 Normal Acid-Base Balance Values

| Value          | Mean variations in normal infants ageing over 7 days |
|----------------|--|
| pH             | 7.42-7.45  |
| $\text{PCO}_2$ | 30-35 mm Hg  |
| BE             | from -3.2 to -0.65 mmol/l                            |
| BB             | 32-46 mmol/l   |
| SB             | 20-24 mmol/l   |
| AB             | 18.5-22.5 mmol/l                                     |

**Acid-base balance as related to anaesthesiology and intensive therapy.** The acid-base balance is regulated by the respiratory system, whose response is very quick (within a few minutes), and the renal function, whose response is slow (pathological changes in the body can be fully compensated by the kidneys within a few days).

When conducting artificial lung ventilation during anaesthesia or for some pathological condition, it is necessary to select thoroughly the ventilation rate because sharp changes in ventilation of the lungs cause significant changes in the acid-base balance of the body. Rapid infusion of fluids during elimination of hypovolaemia may provoke acidosis, and using strong diuretics, which promote a rapid loss of

liquid, can cause alkalosis. Although alkalosis can rapidly be compensated by the respiratory function, it is necessary to remember that even transient abrupt variations in the acid-base state of the body can be extremely dangerous to some patients. The administration of great amounts of fluids for elimination of acidosis often fails to give the desired effect unless the appropriate pathogenetic treatment of acidosis is conducted, while the excretion of these substances by the kidneys may be difficult. Retention of sodium (taken with bicarbonate) or tris-buffer can cause a pronounced pathological iatrogenic effect.

### AGE AND METABOLISM

**Body composition** The human body is a complex structure comprising many organs and tissues. The chemical composition of the body, the relative concentrations of various substances and elements vary substantially with age. For example, the proportion of water or fat in a neonate is higher than in older children or adults, while the proportion of proteins is lower. The amount of fat in a healthy infant born at term is 26 per cent of the body weight, while in children at their prepubertal age it is only 10-11 per cent. The muscles of a neonate are 25 per cent of the body weight, while in an adult male they are 43 per cent. The composition of the body after termination of the pubertal period varies depending on certain endocrine factors. The composition of fat in a child differs from that in an adult: palmitic and palmitoleic acids prevail in them to account for the high melting point of fat. Oleic acid prevails in the fat of elder children and especially in adults. This should be borne in mind when using anaesthetics and medicinal preparations that are readily soluble in fat.

**Metabolism of amino acids and proteins** The differences in the metabolism of amino acids and proteins are mainly associated with age and growth of the body. The daily protein demand in infants is 2.4 g/kg during the first three months of life, 1.85 g/kg at the age of 3-6 months, 1.5 g/kg at the age to 12 months, and 1 g/kg at the age from 1 to 4 years. These amounts satisfy the protein demands (standard protein of milk and eggs). The daily protein demand of a child from 7 to 13 years of age is 0.75-0.9 g/kg, while of an adult, 0.6 g/kg. If the taken protein is of lower value (incomplete set of amino acids, e.g. vegetable protein), its overall amount should be increased accordingly.

In addition to the nine essential amino acids, histidine is also considered to be indispensable for infants under 1 year of age. Methionine cannot convert into cysteine and taurine in premature neonates and these two amino acids are, probably, indispensable for them. Essential amino acids should be about 35 per cent of the 2 g of proteins taken by infants per kg their body weight. This should be 20 per cent for adults. The demands of children ageing

from 1-2 to 10-12 years for separate essential amino acids are not known

The amount of amino acids spent for energy in normal conditions is 4 per cent of the overall energy requirement of neonates. These demands in nurslings increase to 7 per cent and in adults, 13-14 per cent of the total energy produced in the body by oxidation of amino acids.

The amino acids that are not involved in the synthesis of proteins are oxidized to carbon dioxide and water with liberation of energy and nitrogen, which is involved in the synthesis of ammonia and urea, the final product of protein (amino acid) metabolism. Urea is mainly excreted by the kidneys. The nitrogen of urea is about 85 per cent of the total amount of nitrogen contained in the urine of neonates during their first three days of life. This amount decreases to 60 per cent from the 4th or 5th day to the age of 2 months. In adults, the urea nitrogen is 80 per cent of the total nitrogen excreted with the urine. The remaining amount of nitrogen is contained in creatinine, amino acids, whole protein, ammonia, uric acid, and other nitrogen-containing substances.

**Carbohydrate metabolism** The carbohydrate metabolism of children does not substantially differ from that of adults. The main differences reside in the absolute and relative amounts of the metabolized carbohydrates, in the concentrations of substrates and intermediary metabolites in the blood serum. Neonates and infants are less sensitive to decreased concentration of glucose in blood. The symptoms of hypoglycaemia in neonates become obvious with glucose concentrations below 2.22 or 1.67 mmol/l (40 or 30 mg%) and sometimes even 1.11 mmol/l (20 mg%). Normal glucose concentration in the blood is given in Table 12.

The rate of fractional elimination of glucose from the blood in healthy children during glucose tolerance test is comparable with that in adults.

The proportion of energy produced from carbohydrates by breast-fed infants is 38 per cent, while in adults this percentage increased to 58 per cent. The carbohydrate demand in infants, as related to the body weight, is two times greater than in adults. This demand related to the body surface is the same for adults and neonates: 40-42 g glucose per square metre of the body surface.

**Fat metabolism** The intermediary lipid metabolism of children (like all other types of metabolism) does not differ from that of adults. Like in other cases, the differences are mainly quantitative. Substantially different are the first stages of fat digestion in infants. The emulsifying activity of bile acids is low in neonates, and the activity of the gastric lipase is therefore important. This should be borne in mind when giving unemulsified fats to the neonates through a gastric tube. The gastric phase of digestion is indispensable



Table 12 Normal Concentrations of Glucose and Some Intermediary

| Substance | Neonates<br>(1-3 hours) | 4 hours   | 15 days   | 1-12 months | 2 years   |
|-----------|-------------------------|-----------|-----------|-------------|-----------|
| Glucose   | 4.44±1.11               | 2.78±1.39 | 3.61±1.11 | 3.61±1.11   | 3.77±1.11 |
| Lactate   | 2.0-2.4                 |           |           | 1.3-1.8     |           |
| Pyruvate  | 0.17-0.32               |           |           | 0.06-0.13   |           |
| Citrate   | 0.026-0.286             |           |           | 0.099-0.156 |           |

in such infants. This phase may only be excluded on condition that special food is given, which does not require complete processing by the intestinal enzymes. Part of nutrients can otherwise remain not assimilated.

Under normal conditions, fat consumption per kg body weight of a child is much higher than in adults. In an infant it is 7-7.5 times higher, in a 1-year-old child it is 6 times, and in an infant, 3-4 times higher than in an adult. The usual daily fat demand of an adult is 1 g per kg body weight. Fat consumption per unit body surface in children is also higher than in adults (about 1.5-4 times higher).

The concentration of the main intermediary lipid metabolites in children is either the same as in adults or slightly increased. The main differences are observed in neonates during their first seven days of life and in children of other ages. This is explained by the post-stress metabolism in neonates during their adaptation to the extrauterine life. The concentration of non-esterified fatty acids, the ketone bodies, glycerol, and triglycerides of blood serum increases during this period, and the respiratory coefficient changes significantly. This indicates the predominant utilization of fat in the infant body. Age differences in the blood cholesterol and also the esterified to non-esterified fat ratio are also observed.

Tolerance of fat (administered intravenously as emulsion) is high in children, especially in infants. The results of intravenous fat tolerance test are practically always higher in children than in adults. It is possible therefore to suggest that fat metabolism, fat tolerance, and fat demands are higher in children (especially in infants). This can probably be explained by the higher intensity of energy metabolism (as related to the body weight), by the higher lipid demands for the production of energy and by some other anatomical and physiological features of children.

**Energy metabolism.** One of the main aspects in energy metabolism of man is basal metabolism. Basal metabolism is the amount of energy which should be released by the body to maintain the vital processes in a fasting living body at rest at an ambient temperature of 20°C and in the absence of subjective feeling of cold or warmth. The rate of basal metabolism (as related to body weight) varies in

## Metabolites in Blood (mmole/l)

| 4 years   | 6 years  | 9 years   | 12 years  | Adults  |
|-----------|--|-----------|-----------|---|
| 4 00±1 11 | 4 27±1 11<br>0 89-1 7<br>0 05-0 13<br>0 099-0 0156 | 4 05±1 11 | 4 55±1 11 | 4 44±1 11<br>0 67-1 8<br>0 05-0 13<br>0 088-0 167 |

neonates during their first days of life between 40 and 50 kcal/(kg × day), while by the end of the second week of life it increases to 50-55 kcal/(kg × day). The basal metabolic rate of premature neonates is slightly lower than in neonates born at term. The rate of metabolism may increase 2-3 times when a nursling (especially a neonate) cries. When the neonatal period ends, the basal metabolism (as related to the body weight) gradually decreases to attain 24 kcal/(kg × day) to the age of 21. When an infant grows to the age of about 3 years, the basal metabolic rate (as related to the body weight) decreases by 2.2 per cent per kg weight gain.

Changes in the basal metabolic rate as related to the unit surface of the body have another character. The metabolic rate of a neonate after the adaptation period is about 700 kcal/(sq m × day), by the age of 2-3 years it increases to 1200-1500 kcal/(sq m × day), and by the age of 10 it decreases to 1100 kcal/(sq m × day). This rate in adults varies between 950 and 1050 kcal/(sq m × day).

The basal metabolic rate changes during various pathologies. It increases by 25-30 per cent after surgical operations. In sepsis and peritonitis the basal metabolic rate increases by 50 per cent, in severe skeletal injuries by 50-60 per cent, and in burns by 100 per cent. A 1°C increase in body temperature intensifies basal metabolism by 12-13 per cent.

The specific features of basal metabolism associated with age are connected with the growth of the infant and relative changes in the energy released during oxidation of various substances. Fat is the main source of energy in early childhood. It is about 55 per cent of the total energy released by the body. This value decreases gradually to 40-30 per cent in adults. The ratios of the body weight to the body surface, and of the weight of muscles and fat to the weight of the body are constantly changed in children to account for the said changes.

**Thermoregulation** Thermoregulation is inadequate in infants. The inadequacy is characterized by decreased capacity to physical thermoregulation, which is connected with a certain weakness of the thermoregulating centre, vasomotor reactions, and by practical absence of sweat glands function in infants during their first 45 days.

of life On the other hand, the ratio of the body surface to its weight is two times greater in neonates than in 10-year-old children, and three times greater than in adults It has already been said that relative amount of muscles in infants is much smaller than in older children or adults, while the muscles are the major producers of heat in the body In connection with these special properties of infants, their heat loss through radiation is much greater than in adults or older children Despite the absence of function of sweat glands, the perspiration in infants is quite intense Since the relative surface area of infant body is large, much energy is lost due to evaporation of water from the skin Brown fatty tissue, which is mainly found in the interscapular region, performs the heat-generating function of muscles in neonates Heat formation in the brown fatty tissue is intense Neonates can thus easily become cold or overheated This holds true for premature neonates in particular, because the relative content of fat, especially of brown fatty tissue, and muscles is low in them The proportions of an infant body remain 'unfavourable' by the end of the first year of life, but the thermoregulation can be considered satisfactory at this age

The thermoregulating processes can change rapidly and more intensively in infants than in adults during pathologies This should be remembered when treating children with severe diseases, especially in severe climatic conditions (at high ambient temperature and humidity, in particular)

## Chapter 4

### Assessing the Condition of Vital Functions of an Infant

#### DETERMINING THE CONDITION OF THE NERVOUS SYSTEM

The assessment of the neurological state of a child begins with a thorough analysis of the anamnestic data It is necessary to find out if the neonate has been borne into a normal family or if some other members of the family suffer from congenital diseases, if the pregnancy, labour, peri- and postnatal periods were normal or pathological

*Clinical assessment* of the neurological state of a child includes assessment of consciousness, adequacy of behaviour, motor activity, and the amount of movements Hypertonus, strain, twitching, and excitation in response to stimulation should be assessed It is necessary to check the presence of normal and pathological reflexes It is important to see if the fontanelle is not protruded, and there are no 'finger impressions' on x-ray pictures of the skull

*Electroencephalography* is important for diagnosing both primary and secondary affections of the brain. The diagnosis is based on deviations from normal EEG or the appearance of new elements in it. EEG findings should be compared with the clinical picture. Deviations from normal EEG can be diffuse or focal. Diffuse changes are manifested by slowing down of the wave spectrum and also by a series of peak-waves. Focal changes are slowing and inhibition of the waves and peaks related to the high frequency alpha or beta spectrum. It should be remembered that abnormalities can be seen in healthy children as well. Focal peaks in the state of wake are found in about 2 per cent children ageing under 15 years. The prognosis of the neurological condition on the basis of EEG findings should, therefore, be done very carefully by comparing the findings with the character of the clinical picture.

*Echoencephalography* is a simple method giving information on the intracranial relationships (displacement of the medial structures, dimensions of the lateral ventricles, etc.). Ultrasound studies help the examiner to locate a pathological focus and to assess its size.

*Lumbar puncture* is a clinical method for examination of the nervous system. This is usually done between the 4th and 5th lumbar vertebrae. The child is placed on his side with the thighs flexed on the abdomen. The operation should be strictly aseptic. If the liquor's discharge is spontaneous, its pressure is measured using a special tubular extension. Normal pressure of the cerebrospinal fluid varies within a broad range. Pressures exceeding 200 mm H<sub>2</sub>O are considered to be high. To prevent displacement in the presence of high cerebrospinal pressure, only 2 ml of cerebrospinal fluid should be taken. In case of occlusion or decreased pressure of the cerebrospinal fluid, liquor specimens can only be taken with aspiration. Normal findings during laboratory examination of the cerebrospinal fluid and results of the Pandy test are given in Tables 13 and 14.

Table 13 Characteristics of Normal Cerebrospinal Fluid

| Number of cells in ml | Total protein, mg/l            | Albumin, mg/l | Globulin, mg/l | Sugar mmol/l |
|-----------------------|--------------------------------|---------------|----------------|--------------|
| 1-10                  | 160-240 (200-1000 in neonates) | 140-180       | 20-60          | 2.78-4.44    |

The described methods can be used to assess the condition of the central nervous system in both emergency cases and for planned treatment. Time permitting, ultrasound scanning, pneumoencephalography, and angiography can also be used for establishing a diagnosis.

Table 14 Pandy's Test Assessment

| Reaction    | Albumin content, mg/l |
|-------------|-----------------------|
| Negative    | under 400             |
| Opalescence | 400-500               |
| +           | 500-1000              |
| ++          | 1000-3000             |
| +++         | 3000-5000             |
| ++++        | above 5000            |

### DETERMINING THE CONDITION OF THE RESPIRATORY SYSTEM

The diagnosis and correct assessment of gravity of respiratory insufficiency in a child are only possible by comparison of the clinical picture with laboratory findings and functional tests. The physician must be able to interpret correctly the clinical symptoms of acute respiratory insufficiency. He must also be aware of the diagnostic value and possibilities of special methods of examination.

An important clinical symptom of respiratory insufficiency is *dyspnoea*. It can be inspiratory, associated with obstruction of the upper airways, or expiratory, connected with bronchial impatency. Pronounced tachypnoea is usually characterized by involvement of the accessory muscles in the respiratory act. Tachypnoea associated with atelectasis, pneumonia, or other restrictive processes in the lungs is usually characterized by a markedly decreased depth of breathing. It should however be remembered that dyspnoea can also be connected with pathology of the cardiovascular system, nervous system, etc. But the respiratory insufficiency is not always accompanied by dyspnoea.

*Cyanosis* is a common sign of hypoxaemia. But the degree to which this symptom may be pronounced depends on the haemoglobin content, lighting in the room, and the skin colour, and it cannot therefore be used as a criterion of severity of hypoxaemia.

*Hypercapnia* is characterized by dilatation of the peripheral vessels, increased arterial pressure, hyperhidrosis, and depressive states of various gravity.

*Percussion and auscultation* of the lungs and also chest x-ray provide valuable information about the character and localization of the pathological process. A complete understanding of the causes and gravity of respiratory insufficiency can, however, be only attained by comparing the clinical data with laboratory findings and functional tests.

The characteristics of the respiratory function are arbitrarily divided into three groups: external respiration and mechanical prop-

erties of the lungs, pulmonary gas exchange, and gas composition of the blood. The external respiration and mechanical properties of the lungs are usually assessed by spirometry, pneumotachography, pneumography, and body plethysmography.

*Spirometry* is used to determine and record graphically the tidal and minute ventilation volume of the lungs, vital lung capacity (and its minor subdivisions), oxygen consumption, etc. The test can be conducted with breathing pure oxygen or atmospheric air. Spirometers of any type can be used with older children, while special apparatus should be used for testing neonates.

*Pneumotachography* is the recording of the flow rate of respired gas. It can be used for the same purpose as spirometry except for the determination of oxygen consumption. Pneumotachography can also be used to determine the amount of non-elastic breathing resistance (with interruption of the gas flow).

*Pneumography* is the method for studying the respiratory movements. The chest movements are recorded by means of electrodes (impedance pneumography) and sensors which are attached to the patient's chest, the frequency and depth of respiration of the patient can thus be recorded for lengthy periods of time without disturbing the patient.

*Body plethysmography* in combination with impedance pneumography are used for recording frequency and volumetric indices of the external respiration, and also for quantitative assessment of the aerodynamic resistance of the airways. Synchronously taken records of pneumo- and barograms are used for plotting the respiratory loop whose area is proportional to the work done to overcome the aerodynamic resistance of the airways.

Early signs of increased work of breathing associated with increasing aerodynamic resistance and other causes are important manifestations of respiratory dysfunction. Unfortunately, this dysfunction is not revealed in due time because an infant can compensate hypoxaemia and hypercapnia for a certain period of time by intensifying the work of breathing. When the compensatory mechanisms are exhausted, drastic changes in gas exchange become obvious. Increased work of breathing can be determined by pneumotachography and body plethysmography.

The clinical signs of increased work of breathing are the forced posture of the child in its bed, involvement of the accessory muscles in the respiratory act, retraction of the yielding parts of the chest, dilatation of the nares, etc.

The *pulmonary gas exchange* is usually examined by various quick-acting gas analysers. Dead space, the alveolar ventilation, the share of the venous efficiency, the alveolar-arterial gradient, and diffusion capacity of the lungs are of great clinical importance. Radioisotopic methods of examination give a more accurate picture of pulmonary

ventilation and perfusion and their relationships. The acid-base balance and gas content of blood are very important for assessment of the respiratory function.

### DETERMINING THE CONDITION OF THE CARDIOVASCULAR SYSTEM

A constant control of the cardiovascular system is a prerequisite condition in paediatric anaesthesiology. Feeling the pulse, listening to the heart sounds, and determining the condition of microcirculation are the simplest clinical methods to assess haemodynamics.

*Pulse is palpated* to reveal some changes characterizing the action of the heart and vessels (pulse rate, rhythm, filling, pressure, etc.). The pulse is usually felt on the arteries of the extremities, most commonly on the radial artery. Pulse filling gives information on the amount of arterial pressure and blood flow rate. Comparison of the pulse rate at the periphery with the heart rate reveals pulse deficit.

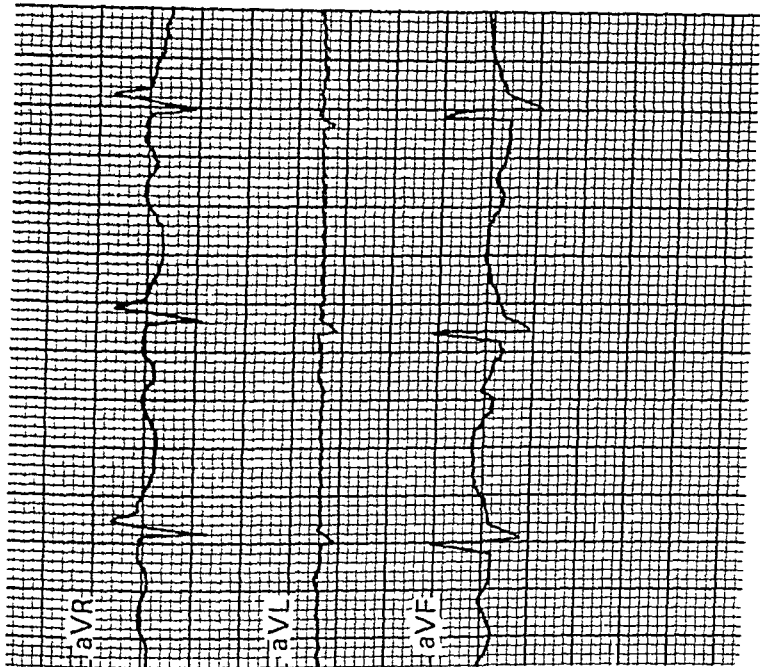
*Auscultation of the heart sounds* is very important diagnostically. It has been established that dulled heart sounds usually precede heart failure. When giving anaesthesia, the sensitive element of the stethoscope is usually fixed with adhesive tape on the heart beat area, an intraoesophageal stethoscope is used during operation on the thoracic organs.

The *condition of microcirculation* is assessed in the clinical practice by the speed with which the capillaries are filled with blood. Pressure is applied to the end of the nail (ear lobe, skin of the forehead, forearm, etc.) until it becomes white. The pressure is then released and the rate of blood filling in the capillaries is determined. The rate of capillary filling is markedly slow in the presence of spasm of arterioles (shock or hypovolaemia).

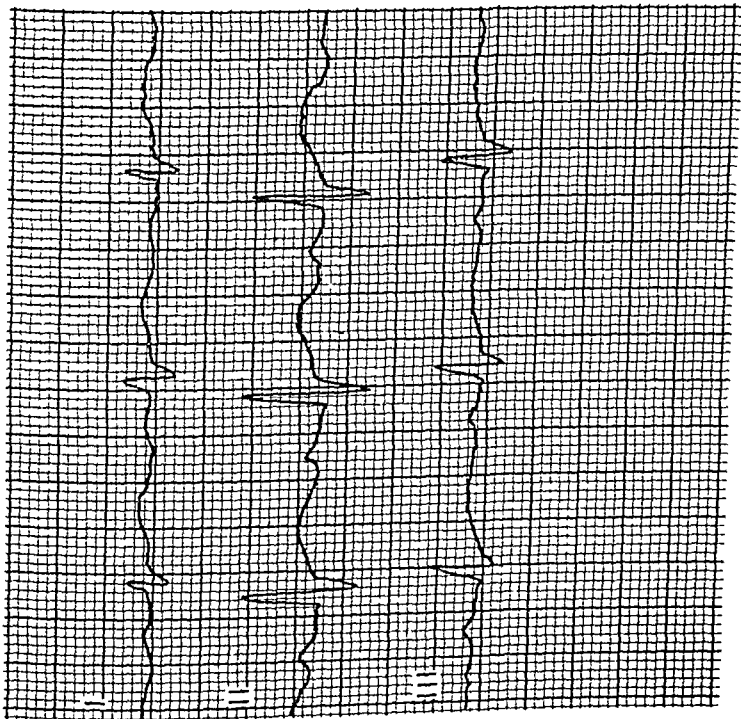
*Arterial pressure* is an important characteristic of blood circulation. The breadth of the cuff used for measuring arterial pressure by the Korotkov method depends on the age of a child: it is 3 cm for neonates, 5 cm for nurslings, and 7 cm for infants ageing from 2 to 7. When arterial pressure is measured by palpation, it should approximately be equal to systolic pressure. Diastolic pressure cannot be measured by this method.

Fig 21 Changes in ECG

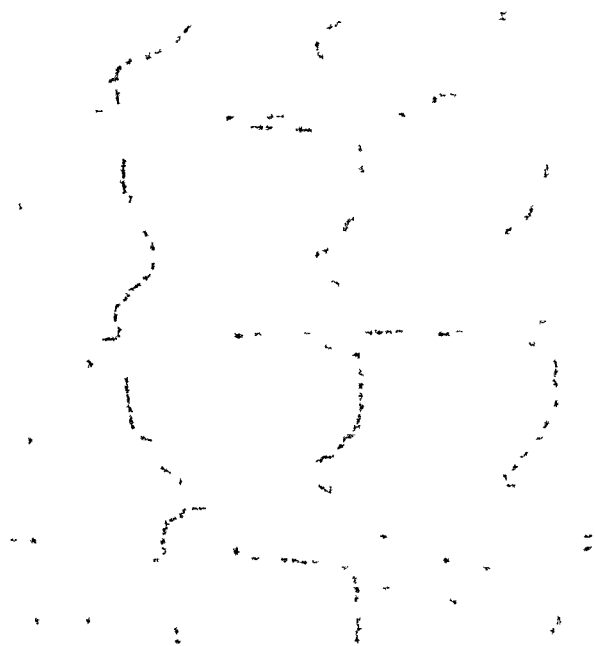
(a) hypokalaemia *T* wave is levelled, *U* wave appears, (b) hyperkalaemia *T* wave sharpens and increases in height, the *QRS* complex is distorted and broadened, the *ST* segment is depressed, (c) sinus node insufficiency, sinus bradycardia, intra-atrial migration of pacemakers, sino-atrial block (lead II continuous record)

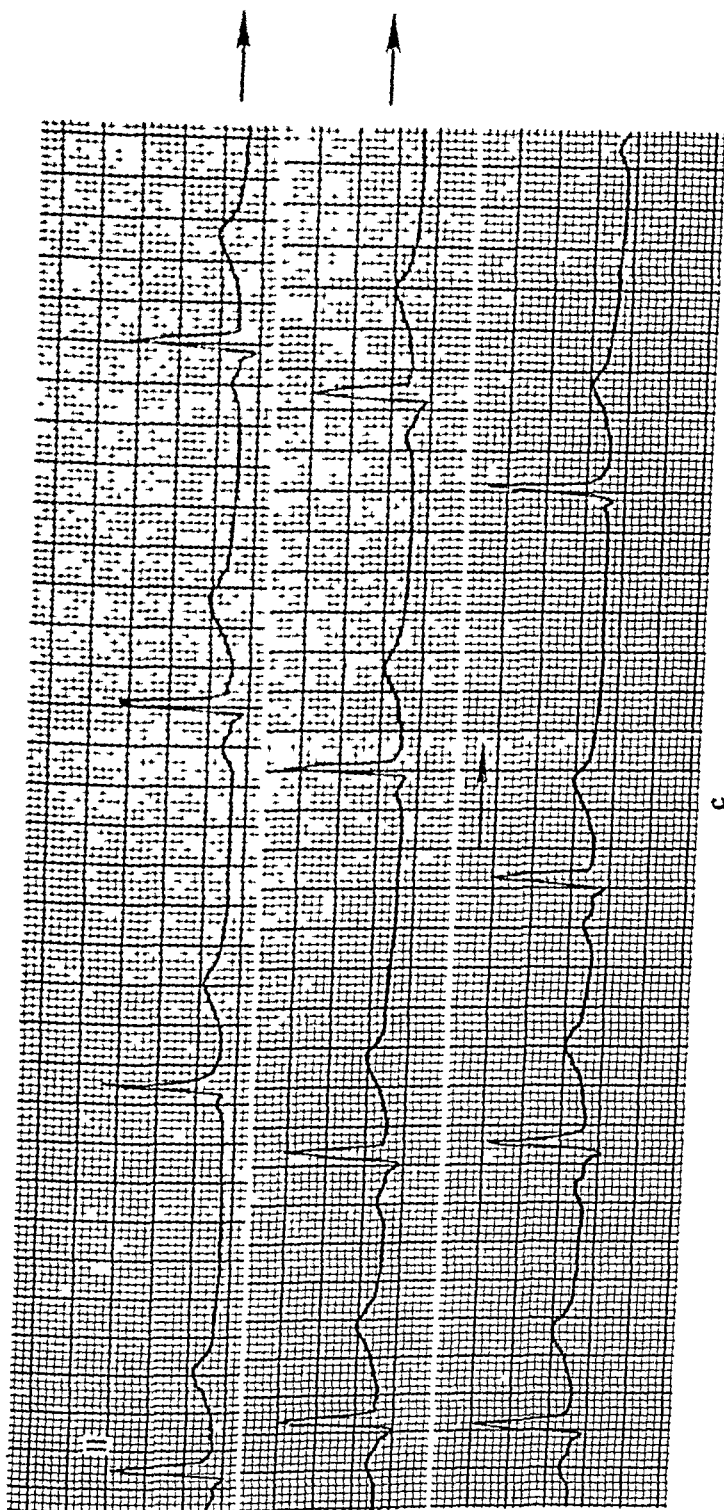


a









Most accurately arterial pressure can be measured by the direct method. A tube is passed in an artery (better the radial artery) and the sensor of the measuring apparatus is connected to the tube. Modern monitors give digital reading of systolic, diastolic and mean pressure. Moreover, a pressure curve is drawn on an oscillograph.

*Central venous pressure* is a very important characteristic of the cardiovascular function. It can be measured by catheterization of a central vein. Valdman's phlebomanometer is used for taking venous pressure. The central venous pressure and its variation during infusion of fluids are very important means of assessing the volume and reserves of the myocardium.

*Electrocardiography* is widely used in clinical physiology because of its relative simplicity. It is used to diagnose disordered automaticity, excitability, and conduction of the myocardium. Electrocardiography is very helpful in diagnosing cardiac arrhythmias, focal and diffuse myocardial affections. The specific ECG changes in hypertrophy of various heart chambers help diagnose congenital and acquired heart diseases. The upset balance of some plasma electrolytes (for example, potassium disturbance) also becomes obvious from ECG changes (Fig. 21). Synchronous recording of electro-, phono-, and sphygmograms (*polycardiography*) gives information about the contractile power of the myocardium and changes in the pulmonary blood circulation in norm and pathology.

Determining the *cardiac output* provides the most comprehensive information on the condition of the cardiovascular function. The Cardio-Green dilution method is often used in paediatric practice for this purpose. The dye is introduced through a catheter into the vena cava or the right atrium, while the dilution curve is recorded by means of an apparatus whose pickup is attached to the ear lobe. Knowing the cardiac output, it is easy to calculate the stroke volume, the work of the ventricles, the systemic vascular resistance, and other important haemodynamic characteristics.

Among methods to assess blood circulation without operative intervention, tetrapolar rheography and the method employing the Doppler effect should be mentioned.

## DETERMINING THE WATER-ELECTROLYTE METABOLISM

The water-electrolyte metabolism in children is assessed by the anamnestic data, clinical signs of pathology, and laboratory findings. When collecting anamnesis, special attention should be paid to uptake and excretion of water by the child because this information can suggest some conclusions. The clinical signs of upset water-salt metabolism are pronounced changes in the body weight occurring within short periods of time (to 3 days), changes in the turgor and elasticity of the skin, among the clinical signs in infants are the

condition of the fontanelle, eye-ball convergence, the condition of the mouth mucosa, the amount of the urine excreted, its colour, the central and peripheral blood circulation, and the action of the central nervous system

The laboratory findings of special importance are the haematocrit, the concentration of haemoglobin and of the main ions (sodium, potassium, chloride calcium, and sometimes magnesium) in the blood serum and in erythrocytes (sodium and potassium), diuresis, specific gravity of the urine, the sodium and potassium content of the urine, concentration of protein and urea in the blood serum, and osmolarity of the blood and urine

The arterial pressure, the central venous pressure and the pulse rate may also be important for diagnosing disorders in the water-electrolyte metabolism of a child during clinical and instrumental examinations

**Types of disordered water-electrolyte metabolism in children.** Two major disorders in the water-electrolyte metabolism are distinguished dehydration (the reduction of the overall amount of water in the body) and hyperhydration (increased total amount of water in the body) These major disorders are each subdivided into three groups of disorders in which the degree of disorder is expressed in terms of concentration or tonicity (osmolarity) of the extracellular fluid isotonic (normal tonicity or osmolarity of the extracellular fluid), hypotonic (subnormal osmolarity of the extracellular fluid), and hypertonic (increased osmolarity of the extracellular fluid) The type of disorder is determined accurately by laboratory testing

The degree of disorder in the water-electrolyte metabolism is determined by the relative amount of change in the body weight to either side The disorder of degree I in infants is characterized by the change in the body weight of 5 per cent, degree II is characterized by the change to 10 per cent, and degree III is characterized by the change in the body weight to 15 per cent In children ageing over 3-4 years these figures are 3, 6, 9 and 10 per cent, respectively (as measured with respect to the body weight before the disease) A loss of water by an infant exceeding 15 per cent of its body weight (10 per cent in children over 3-4) is often a critical condition When assessing the body weight deficit it is necessary to consider a possible subacute or even a chronic disease which may account for the deficit The quantitative and qualitative characteristics of nutrition are important for the anamnesis of the disease, especially so if the disease lasts more than 3-4 days a child may lose 1 per cent of its body weight a day if it does not eat Disregarding these factors may be the cause of erroneous conclusions

Table 15 gives the main diagnostic criteria to assess the type of dehydration A correct conclusion can be drawn after laboratory testing Except in very few diseases, hyperhydration is usually

caused by iatrogenic factors, due to inappropriate medication as a result of incorrect assessment of the water-electrolyte metabolism of the patient. The main signs of hyperhydration are an abrupt increase in body weight, development of oedema, and partly enlargement of tissues. Oedema usually occurs when the body weight increases by more than 5 per cent. The cardiovascular system is often involved. The specific symptoms of hyperhydration are absent (except if its degree is high). Types of hyperhydration are not differentiated clinically. Their differentiation is based on determining the sodium concentration or osmolality of the serum.

Table 15 Clinical and Laboratory Diagnosis of Dehydration Type

| Criterion               | Isotonic  | Hyperosmotic  | Hypoosmotic  |
|-------------------------|---|---|--|
| Condition               | Medium gravity  | Satisfactory  | Good and critical  |
| Skin turgor             | Decreased   | Normal or decreased   | Markedly decreased   |
| Mucosa                  | Dry   | Very dry  | Moist, excretory   |
| Eye-balls               | Retracted   | Normal  | Markedly retracted   |
| Fontanelle (in infants) | Retracted   | Retracted   | Retracted  |
| Temperature             | Normal  | Elevated  | Subnormal  |
| Arterial pressure       | Normal or decreased   | Normal  | Hypotension, often pronounced  |
| Central venous pressure | Low   | Normal or low   | Depends on heart function  |
| Diuresis                | Decreased   | Very low  | Increased  |
| Sodium of serum         | 135-145 mmol/l  | 145-150 mmol/l  | 135-130 mmol/l   |
| Haematocrit             | Increased   | Increased   | Increased  |
| Serum protein           | Increased   | Increased   | Increased  |
| Osmolality of serum     | Normal  | Increased   | Decreased  |
| Urea of serum           | Increased   | Increased   | Increased  |
| Sodium of urine         | Decreased   | Increased   | Decreased  |
| Anamnestic data         | Recurrent vomiting with moderate volumes of gastric content, stools are liquid and frequent | Elevated body temperature, dyspnoea, sweating, less frequently vomiting | Recurrent vomiting with large volumes of contents of the stomach and the upper portions of the small intestine, stools are watery and frequent, fistula in the small intestine |

**Disorders in the ion metabolism.** Ion metabolism (like in water exchange) becomes upset in the presence of imbalance between uptake and excretion of one or several ions. Four types of upset ion metabolism are distinguished: decreased or increased total amount of a particular ion in the body, increased or decreased concentration of the ion in body fluids. Mixed-type disorders are frequent too. In most cases the upset ion metabolism is associated with upset water metabolism. Upset metabolism of only one or several ions occurs in rare cases. In most cases the metabolic disorders are due to incorrect treatment, less frequently they are a specific feature of a disease. Accurate diagnosis of the type of metabolic disorder is difficult but possible. It is necessary to determine the ion concentration in the extra- and intracellular fluids, the volumes of fluid in the extracellular and intracellular spaces, and the ion balance (the difference between uptaken amount of the ion and its excretion by all possible routes). This information enables one to determine the total ion content of the body, the tendency to changes in the balance, the concentration of the ion in the extra- and intracellular fluids, and also their proportions. This is an important information for planning a correct treatment.

The changes in the total ion concentration in the body and the intracellular fluid are usually characterized as changes in the concentration of a particular ion in the blood plasma (in the serum and the extracellular fluid), as is the case with hypo- or hypernatraemia, hypo- or hyperkalaemia, etc. When diagnosing ion metabolic disorders, it is necessary to determine thoroughly the water metabolism because these two types of metabolism are closely inter-related.

Potassium is the main ion of the intracellular fluid, which contains 98 per cent of the total potassium contained in the body. This ion is involved in many physiological and biochemical processes occurring in the cell. The extracellular fluid contains only 2 per cent of the total potassium. The gradient between the intra- and extracellular potassium ion is directed toward the extracellular fluid. This gradient is maintained by about 80 per cent of the energy released by the cell.

Among disorders associated with metabolism of sodium and potassium ions is transmineralization. This is a simultaneous redistribution of the sodium and potassium ions between the extra- and intracellular fluids in opposite directions without alteration of their contents in the body but with alteration of their concentrations in the extra- and intracellular fluids. Transmineralization occurs in cases with decreased activity of the potassium-sodium pump and may be caused by any factor interfering with the energy release by the cell.

The total reduction of the potassium content in the body can be caused by the negative balance of the ion even for a short period of

time (2-3 days, and sometimes even during one day) The clinical picture of the potassium deficit in the body is quite specific flaccidity, adynamia, muscular hypotonia, decreased motor activity of the intestine (to a complete paresis), tachycardia, worsened contractile capacity of the myocardium, enlargement of the heart, and energy-dynamic insufficiency determinable on ECG

Laboratory tests show decreased potassium in the erythrocytes and later in the plasma (*hypokalaemia*) Hypokalaemia can be clearly seen on ECG as increased voltage of the *P* wave, low *R* wave, and flattening and broadening of the *T* wave (see Fig 21, *a*) The potassium deficit is attended by alkalosis The general potassium deficit is thus characterized by the general decrease of the total cation in the body and its concentration in the extra- and intracellular fluids It should be noted that the potassium content of the blood serum sometimes remains normal, while the deficit already exists along with clinical symptoms and a permanent tendency to decreasing potassium concentration in the erythrocytes

*Hyperkalaemia* is the upset potassium metabolism characterized by increased potassium concentration in the blood serum This condition often occurs in the presence of upset excretory function of the kidneys It should be noted that cases with increased total concentration of potassium in the body have not been reported, and by hyperkalaemia is therefore understood only increased potassium concentration in the extracellular fluid Hyperkalaemia can develop in the presence of acidosis and dehydration

### DETERMINING THE ACID-BASE BALANCE

The assessment of the acid-base balance and analysis of gases in the blood are obligatory for examination of patients in order to assess the severity of respiratory disorders and to conduct intensive therapy The clinical assessment of the acid-base state includes the determination of pH,  $PCO_2$ ,  $PO_2$ , and BE

Disorders of the acid-base balance are classified as alkalosis and acidosis (Fig 22) Both alkalosis and acidosis may be respiratory and metabolic Acidosis is the active reaction of the body medium, the condition in which the hydrogen ion concentration exceeds the permissible limit The pH of the medium is thus below 7.35 Alkalosis is characterized by an abnormal decrease of the hydrogen ion concentration The pH of the medium is thus above 7.45

*Respiratory disorders* Respiratory acidosis is the condition associated with lung hypoventilation with the corresponding increase in  $PCO_2$  in the body fluids and accumulation of the hydrogen ions Elevation of  $PCO_2$  above 40 mm Hg indicates respiratory acidosis, which arises as a result of inhibition of the respiratory centre, obstruction of the airways, alveolar hypoventilation associated with

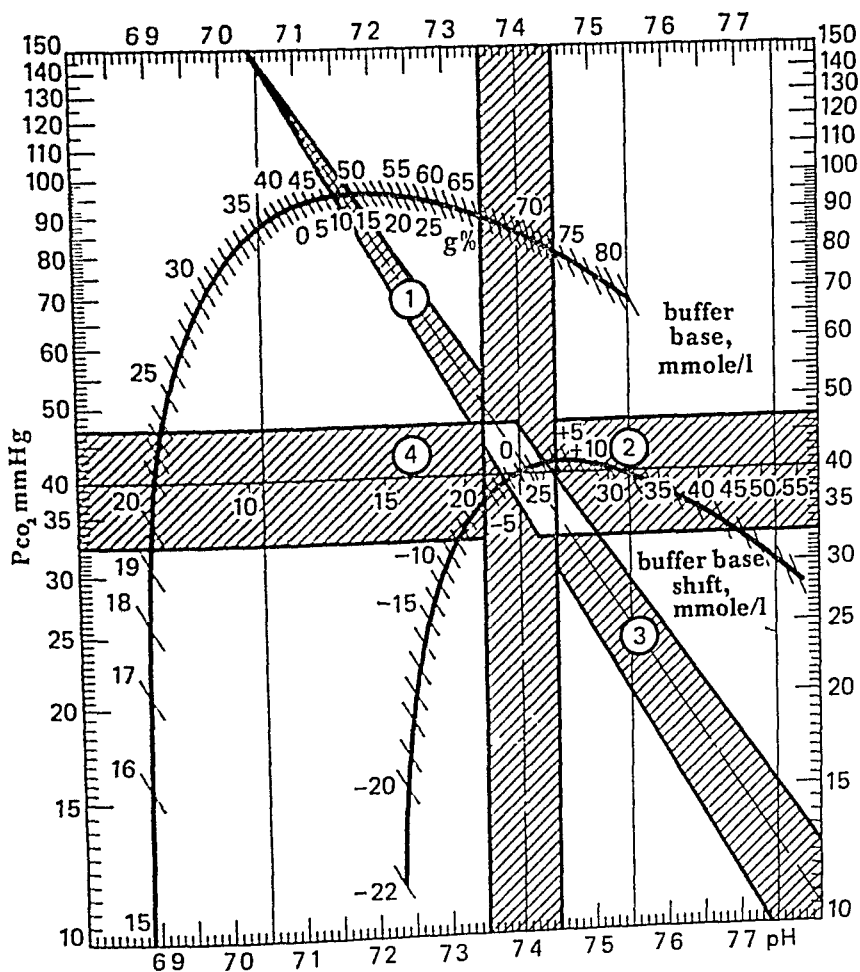


Fig 22 Acid-base disbalance

0—normal acid-base balance, 1—respiratory acidosis, 2—metabolic alkalosis, 3—respiratory alkalosis, 4—metabolic acidosis

diseases of the lungs, and other conditions. Increased  $PCO_2$  associated with metabolic alkalosis is compensatory by its character because  $CO_2$  neutralizes excess amounts of nonvolatile bases.

**Respiratory alkalosis** is caused by lung hyperventilation, decreased  $PCO_2$  in the body fluids, and the respective decrease in the hydrogen ion concentration.  $PCO_2$  below 35 mm Hg indicates respiratory alkalosis due to hyperventilation developing from incorrect artificial lung ventilation or affections of the central nervous system.

**Metabolic disorders** Metabolic acidosis is a common condition associated with acid-base imbalance. It occurs in the absence or decreased oxygen supply to tissues, adaptation to anaerobic oxidation and underoxidation of some substances, which results in accumu-



lation of hydrogen in the body fluids and decreased concentration of the buffer anions. Metabolic acidosis is thus characterized by pH below 7.35 and negative BE values (see Fig. 22). Metabolic alkalosis is acid-base imbalance commonly occurring in post-aggressive states. It is caused by excessive withdrawal of the hydrogen ion from the body or increased concentration of the bicarbonate ion, phosphate and protein ions. Excessive loss of the hydrogen ions occurs during vomiting and after administration of diuretics. The loss of the potassium ion from the body is, probably, the most important mechanism of metabolic alkalosis. It causes transition of the hydrogen ion from the extracellular fluid into the intracellular fluid (with subsequent development of alkalosis in extracellular fluid and acidosis in intracellular fluid) and additional loss of the hydrogen ion with the urine. The latter is connected with the fact that hydrogen and potassium are excreted in exchange for sodium, and thus compete with one another: the excretion of hydrogen increases with potassium deficit. The increase in the content of the buffer ion (mainly the bicarbonate ion) may increase because of the uptake of large quantity of the anion or oxidation of the organic acid ions (lactate, citrate, acetate ions). Metabolic alkalosis is characterized by the pH above 7.45 and positive BE value.

*Hypoxaemia* is determined by the value of  $PO_2$ . Decreased  $PO_2$  is usually connected with upset ventilation-perfusion relationships and blood shunting from the right to the left. Increased  $PO_2$  (*hyperoxia*) is usually the result of excessively high oxygen concentration in the breathing gas during oxygen therapy.

The diagnosis of acid-base imbalance and establishing which shifts are primary and which changes in the acid-base state are caused by the compensatory processes are sometimes very difficult. The answer can only be given after comparison of the clinical picture of the disease and the laboratory findings. Determining the cause of metabolic alkalosis is a difficult diagnostic aspect. It was earlier believed that metabolic alkalosis develops as a result of hypochloro-aemia. It is possible to suggest now that pure hypochloro-aemic alkalosis occurs very rarely, while hypochloro-aemia attends any alkalosis condition. This can be explained by the fact that the increasing concentration of the bicarbonate ion must be accompanied either by increasing concentration of the cations or decreasing concentration of any ion. The first condition is practically unfeasible, while the chloride ion concentration changes more easily. There is a simple formula by which it is easy to check whether or not the concentration of chlorine in the blood corresponds to the given acid-base balance of the body:  $BE + 42 = (\text{approximately}) Na^+ - Cl^-$ . The value of 42 mmol/l corresponds to the concentration of buffer bases (less bicarbonate). When determining metabolic alkalosis and verifying if it is not a compensation of respiratory acidosis,

two other tests should be performed as well the potassium content of the erythrocytes and the pH of the urine should be determined

If alkalosis is associated with decreased potassium content in the erythrocytes, and also with the acid reaction of the urine, one may suggest, with a great degree of certainty, that metabolic alkalosis is the result of potassium deficit in the body

In emergency cases, in order to assess the condition of the respiratory system, it is often sufficient to compare the clinical signs (the appearance of the child, its behaviour, colour of the skin and the mucosa, percussion and auscultation findings) with x-ray findings during the examination of the acid-base state. The above described functional tests may also be conducted during routine examinations.

### ASSESSING MAIN TYPES OF METABOLISM

**Determining changes in protein metabolism.** Changes in the protein metabolism during various diseases requiring surgical treatment or intensive therapy are usually diagnosed by laboratory techniques. The main characteristics are concentration of total protein in the blood serum, of albumin, the albumin to globulin ratio, relative concentration of various globulins in the blood (proteogram), urea concentration, creatinine of serum, various serum amino acids, the excretion of urea or total nitrogen with the urine, the amount of amino acids excreted with the urine, the nitrogen equilibrium as determined by the difference of the nitrogen uptaken and excreted by all possible routes. These findings can be used to assess the condition of the protein metabolism of a child.

**Determining changes in carbohydrate metabolism.** The concentration of glucose in the blood and the urine is usually determined in the clinical practice. Despite the simplicity of the studies, correctly interpreted findings and their comparison with the clinical data can be quite informative for the assessment of the carbohydrate metabolism. Additional data can be obtained during the determination of concentration of intermediary products of the carbohydrate metabolism and by conducting peroral or intravenous glucose tolerance tests, and some other studies.

Hyperglycaemia often occurs in children in critical states and during intensive therapy. It arises as a result of disbalance between the rate of glucose delivery into the extracellular fluid and the rate of its consumption in the peripheral tissues of the body. This condition can be connected with metabolic changes occurring during stress when the activity of endogenic insulin is inhibited and the passage of glucose through the cell membrane is disturbed. These changes can upset the Krebs's cycle to block the entrance of glucose metabolites due to prevalence of fat metabolism. The catecholamine level is also important. Increased content of catecholamines, which

stimulate glycogenolysis, increases the glucose supply to the extracellular fluid. During early sepsis, the action of bacterial endotoxins on the vessels disturbs the peripheral circulation and decreases the glucose consumption in the peripheral tissues. The above described stress reaction of the carbohydrate metabolism can concur simultaneously. Hyperglycaemia can be connected with administration of glucose in doses that cannot be utilized by the child's body. Infusion therapy (the glucose dose and the rate of its administration) should therefore be selected in accordance with the condition of the child and the intensity of metabolism. It should be remembered that hyperglycaemia proper can cause pathological conditions such as hyperosmolarity of the extracellular fluid or osmotic diuresis, which in turn can cause dehydration.

**Determining changes in fat metabolism.** Changes in the fat metabolism are mainly diagnosed by analysing the results of laboratory examination of blood serum. The main signs of upset fat metabolism are changes in the concentration of non-esterified fatty acids, total lipids, cholesterol and its fractions, phospholipids and their fractions, triglycerols, less frequently di- and monoglycerols, some polyunsaturated fatty acids, and their proportions. The concentration of these substances can change due to their insufficient or excessive supply into the blood, with intensification or weakening of intensity of their conversions, and due to some other causes.

**Determining changes in energy metabolism.** The diagnosis of changes in the energy metabolism is based on measuring the basal metabolism by indirect calorimetry and comparing the findings with the standards for children of the appropriate age. Moreover, when determining the degree of protein decomposition by the formation of the end products of its metabolism one can calculate amounts of components of the metabolized mixture during certain periods of time. Comparison of these changes with normal findings shows not only the changes in the energy metabolism but also the metabolism of energy substrates, i.e. glucose, fat and protein.

*Thermal regulation* is closely connected with energy metabolism. The diagnosis of its disorders is very simple. It is only necessary to know the body temperature, the ambient temperature, and air humidity. Knowledge of these parameters is sufficient for the assessment of thermal regulation of a child at a given moment.

## Chapter 5

# Anaesthetics and Other Medicines Used for Anaesthesia, Resuscitation, and Intensive Therapy of Children

### GENERAL ANAESTHETICS

#### Inhalation Anaesthetics

*Nitrous oxide*  $\text{N}_2\text{O}$  ('laughing' gas) is a colourless gas, heavier than air, having no specific odour, with a sweetish taste. A volume of nitrous oxide dissolves in about two volumes of water at  $15\text{--}20^\circ\text{C}$ . 1 kg of liquid nitrous oxide gives 500 litres of laughing gas. The gas is nonflammable but supports burning actively, especially when mixed with ether, cyclopropane, ethyl chloride and some other gases. It does not combine with the haemoglobin and is present in the plasma in the dissolved state. When inhalation of the gas is discontinued, it is withdrawn from the body through the respiratory tract in the unchanged form, all gas is removed from the body in 10-15 minutes. Nitrous oxide is a mild anaesthetic. It is given in a mixture with oxygen. A mixture of 70-80 per cent nitrous oxide with 20-30 per cent oxygen is usually inhaled by the patient through a special apparatus. Nitrous oxide is widely used in modern anaesthesiology, usually in combination with other anaesthetics, e.g. ether, cyclopropane, and others, for neuroleptanalgesia and balanced anaesthesia. When the delivery of nitrous oxide is discontinued, oxygen should be given to breathe for another 4-5 minutes to prevent diffusion hypoxia. Commercial nitrous oxide is usually available in the liquefied form (in metal cylinders).

*Cyclopropane* is a colourless inflammable gas with a specific odour and pungent taste. The boiling point of the gas is  $34.5^\circ\text{C}$  (at normal pressure). When mixed with air, oxygen, or nitrous oxide it forms explosive mixtures which burst from open flame. A volume of liquid cyclopropane forms 376 volumes of gas. Cyclopropane is an effective anaesthetic. It is discharged from the lungs in a practically unchanged form within 10 minutes. It does not affect the liver function. Due to its constricting effect on the renal vessels, the diuresis slightly decreases, thereby increasing the arterial pressure, slowing down the pulse rate, and causing arrhythmia. Cyclopropane increases markedly the sensitivity of the myocardium to epinephrine and may provoke ventricular fibrillation. The gas is used for induction and maintenance of anaesthesia. More commonly cyclopropane is used as a component for combined anaesthesia in combination with nitrous oxide. The gas can also be used together with oxygen.

The common aftereffects of cyclopropane anaesthesia are headache, vomiting, and intestinal paresis.

Liquefied cyclopropane is usually produced for sale in 1 or 2-litre steel orange-coloured cylinders which should be kept away from open flame.

*Anaesthetic ether* is a colourless, transparent, volatile and highly inflammable liquid with a characteristic odour and burning taste. When mixed with air, oxygen, or nitrous oxide in certain proportions it forms explosive mixtures. A litre of liquid ether gives 2.20 litres of vapour. Anaesthetic ether is not used today for induction of anaesthesia but is only used to maintain narcosis. Recovery from ether anaesthesia takes much time (about 20-30 minutes, with subsequent sleepiness and depression for a few hours). The post-operative period (after ether narcosis) is characterized by nausea and metabolic acidosis. Ether anaesthesia is contraindicated for acute respiratory diseases, operations associated with electrocoagulation, severe diseases of the kidneys and the liver.

Ether is available in 150-ml dark glass bottles, which should be kept in the dark and away from open flame.

*Halothane* (fluothane, florotan) is a colourless, transparent liquid with the odour of chloroform and a sweetish burning taste; the liquid is nonflammable. Mixtures of halothane with air, oxygen, or nitrous oxide are not explosive. Halothane is an effective general anaesthetic, much more effective than ether or chloroform. Anaesthesia can be induced by inhaling a mixture containing only 0.02-0.03 l/l (2-3 per cent v/v) of halothane, and maintained by 0.005-0.015 l/l (0.5-1.5 per cent v/v). The anaesthetic acts quickly: the patient becomes unconscious in 1-2 minutes without anxiety. The surgical stage of anaesthesia is attained in 3-4 minutes. The surgical period is characterized by vasoplegia with adequate peripheral blood flow and a hypotensive effect (the pressure decreases by 10-20 mm Hg). The pulse rate usually slows down, the breathing may quicken. The muscles of the neck, the maxilla, and the extremities are relaxed. The length of the recovery period depends on the duration of anaesthesia and averages from 5 to 15 minutes. The post-narcosis depression lasts for 30-60 minutes. Halothane vapour does not irritate the mucosa and dilates the bronchi. Hypotension is partly explained by the inhibiting effect of the preparation on the myocardium, the sympathetic ganglia, and the peripheral vessels. Halothane increases the sensitivity of the heart to catecholamines, thus accounting for possible arrhythmia (to ventricular fibrillation especially after administration of epinephrine and norepinephrine).

In modern anaesthesiological practice, halothane is used in mixtures with oxygen, or nitrous oxide and oxygen, or with ether (azeotropic mixture of two volumes of halothane and one volume of ether). Contraindications for using halothane are severe organic

diseases of the liver Halothane should be used with precautions in cases with hyperadrenalaemia (pheochromocytoma)

Halothane is available in dark 50-ml bottles It should be kept in the dark and dry place

*Chloroform* is a clear colourless liquid with a specific smell and a sweetish burning taste Chloroform vapour is nonflammable or explosive It is a strong narcotic the patient becomes narcotized within 5-6 minutes from inhalation of chloroform in the concentration of 0.03-0.04 l/l (3-4 per cent v/v) Anxiety is not obligatory Anaesthesia is maintained by the concentration of 0.01-0.015 l/l (1.0-1.5 per cent v/v) The patient wakes up in 5-10 minutes, the post-narcotic depression lasts for about 30 minutes Chloroform is highly toxic and is therefore used in rare cases for anaesthetic purposes It can cause dystrophic changes in the myocardium, fat degeneration and cirrhosis of the liver, kidneys, and other vital organs

Chloroform is available in 50-ml vials of dark glass It should be kept away from heat

### Non-inhalation Anaesthetics

*Cyclobarbitol* (fanodorm, hevemal) is a white foamy substance soluble in water and 95 per cent alcohol, the aqueous solutions are readily hydrolysed, decomposed on sterilization Solutions are prepared in aseptic conditions, immediately before use Double-distilled water or an isotonic sodium chloride solution are used for dilution The solution should be used within 4 hours Cyclobarbitol is used mainly as a 1-2 per cent solution for induction anaesthesia The narcotic effect is rapid the patient is asleep without excitation in 30-60 seconds and sleeps for 5-20 minutes It can be recommended for minor operations, in wound dressing, and diagnostic operations, e.g. endoscopy Long cyclobarbitol anaesthesia is not practicable now It should be remembered that even small doses of cyclobarbitol can inhibit the respiratory centre The anaesthesiologist must therefore be prepared to give artificial lung ventilation if necessary Nausea, vomiting, and headache are quite rare aftereffects

The anaesthetic is available in vials containing 1-2 g of dry substance The preparation should be kept in dry, cool place protected from light

*Thiopental sodium* is a dry loose yellowish substance readily soluble in water, the solutions are unstable and decompose on standing, a precipitate is formed on boiling A freshly prepared 1-2.5 per cent solution is used for anaesthesiological purpose The same precautions should be taken as with cyclobarbitol Indications for thiopental sodium are the same as for cyclobarbitol as well The patient becomes narcotized with thiopental sodium quicker than with cyclobarbitol,

but its action is shorter. The preparation has a marked vagotonic effect and this can cause laryngo- and bronchospasm. The presence of sulphur in the preparation accounts for its spasmogenic properties. The preparation is contraindicated for patients with severe hepatic dysfunction, especially in the presence of concurrent hypoxia.

The preparation is available in vials, in 0.5-1 g doses. It should be stored in a cool dry place.

*Hydroxydione sodium* (viadril) is a steroid preparation devoid of active hormone properties. A freshly prepared 2.5-10 per cent solution is used for induction anaesthesia. The preparation (0.2-1 g) is administered rapidly into a large vein with obligatory subsequent injection of a 0.5 per cent novocaine solution or isotonic sodium chloride solution. The sleep is induced in 3-5 minutes and lasts for 30-60 minutes. The preparation has no toxic effect on the parenchymatous organs, respiration, or blood circulation. Decreasing laryngeal and pharyngeal reflexes reduce significantly the consumption of relaxants. The preparation sometimes causes local vascular reactions (pain, phlebitis, skin erythema, disturbances in the venous blood circulation).

The preparation is produced in 0.5 g ampoules. It should be stored in the dark.

*Sodium oxybate* (sodium 4-oxybutyrate) is a substance whose structure is similar to that of human metabolites. The dose depends on the body weight. The minimum dose for induction anaesthesia is 65-75 mg/kg body weight. The patient falls asleep in 10-15 minutes and becomes narcotized in 15-30 minutes. The anaesthesia lasts for 40-120 minutes. When sodium oxybate alone is used, the dose should be increased to 120-150 mg/kg. Anaesthesia can thus be prolonged to 3.5 hours. The post-anaesthesia sleep continues for 2 to 5 hours. Sodium oxybate does not inhibit the function of the parenchymatous organs, it stabilizes the blood circulation, the respiratory function is not inhibited significantly and the patient can therefore breathe spontaneously during anaesthesia.

The preparation is produced in 10-ml ampoules (20 per cent solution). It should be stored with precautions. Formerly, the preparation was produced in powder form and was given per os. The sleep was induced in 15-20 minutes.

*Ketamine* (kalipsol, ketalar) is a short-acting anaesthetic with a strong analgesic effect. It can be given separately or in combination with some other anaesthetics. An intravenous administration of 2-3 mg per kg body weight induces a surgical stage of anaesthesia in 30 seconds. The narcotic effect lasts for 5-15 minutes. If anaesthesia should be prolonged, the preparation can be administered repeatedly since it has no cumulative action. The preparation is administered intramuscularly in a dose of 12-14 mg/kg to neonates and 8-10 mg/kg to older children. After administration of the preparation the arteri-

al pressure is usually elevated by 20-25 per cent, the respiration is not inhibited. The preparation is used for anaesthesia during various diagnostic procedures, in minor surgery, for induction anaesthesia, in shock, during transportation, repositioning, and endoscopic examinations. Post-operative excitation can be relieved by small doses of diazepam or droperidol.

The preparation is available in 10 or 20-ml vials (1 ml contains 5 or 10 mg of the preparation). The vials should be kept under lock.

*Propanidid* (sombrevin, eptol) is a pale-yellow oily liquid, sparingly soluble in water. It is a short-acting anaesthetic, quickly and easily inducing sleep and rapid recovery without prolonged depression, which makes it useful for out-patient application. It is administered intravenously using a large-diameter needle. The dose is from 5 to 15 mg per kg body weight. The preparation is contraindicated in shock, cardiovascular pathologies during the decompensation stage, and haemolytic anaemia. The preparation should be used with precautions in patients with hypertension and disordered coronary circulation.

Propanidid is produced in 500-mg ampoules, complete with 1600 mg of diluent (and 70 mg of sodium chloride). The preparation should be kept in cold.

*Etomidate* (hypnomidate) is another preparation used for intravenous anaesthesia. It is characterized by an original chemical structure and a very broad spectrum of therapeutic effect. It is a short-acting anaesthetic; its effect lasts for 8-10 minutes when given in a dose of 0.3 mg/kg body weight. It is widely used in cardio-surgical patients.

*Althesine* (alphathesine) is a steroid anaesthetic without hormone activity with a broad spectrum of therapeutic effect. When injected intravenously in a dose of 0.07-0.1 mg/kg body weight it acts for about ten minutes. A rapid administration of the preparation can cause respiratory depression.

Etomidate and althesine are used in out-patient conditions.

### Local Anaesthetics

*Procaine hydrochloride* (novocaine) is a colourless and odourless crystalline substance, readily soluble in water and alcohol, bitter to taste. It has a local anaesthetic effect and is widely used for infiltration, spinal, and epidural anaesthesia. When injected into the human body the preparation quickly hydrolyses. When absorbed in the blood, or injected directly into the blood vessel, novocaine has a general anaesthetic effect, inhibits the activity of cholinesterase systems, decreases the production of acetylcholine, has a slight ganglioblocking effect, decreases the spasm of smooth muscles and excitability of the heart muscle, and decreases excitability of the



motor zones in the cerebral cortex. A 0.25-0.5 per cent procaine solution is used for infiltration anaesthesia. According to A. Vishnevsky, 0.125-0.25 per cent solutions are used (creeping infiltration). Conduction anaesthesia is attained with 1-2 per cent procaine solutions. The concentration of the preparation for epidural anaesthesia is 2 per cent (20-25 ml), for spinal anaesthesia 5 per cent solution (2-5 ml).

The preparation is produced in 1, 2, 10, and 20 ml ampoules containing 0.25 and 0.5 per cent solutions. A 20 per cent solution is available in 1, 2, 5 and 10-ml vials. The preparation should be kept in the dark.

*Trimecaine* (mesocaine) is a white or yellowish powder, readily soluble in water and alcohol. It is used for conduction and infiltration anaesthesia. It is a more active and longer-acting anaesthetic than novocaine. The preparation's toxicity is low and it does not produce local irritation of tissues. Trimecaine for infiltration anaesthesia is used in the concentration of 0.25, 0.5 and 1 per cent solutions, for conduction anaesthesia the concentration is 1 and 2 per cent. In order to strengthen and prolong the analgesic effect of the preparation, 0.1 per cent epinephrine solution is added (0.1-0.2 ml per 10-20 ml). Trimecaine is well tolerated by the patients. In hypersensitive patients it may cause headache, nausea, and burning or itching feeling in the wound.

The powder should be kept in well stoppered bottles.

*Tetracaine hydrochloride* (dicaine) is a white odourless crystalline powder. Its local effect is much stronger than that of procaine, but the toxicity is ten times higher. The preparation is used to anaesthetize mucosa of the pharynx, larynx, trachea, for endoscopic manipulations. To do so, a cotton ball is wetted with a 0.5-1 per cent dicaine solution (not more than 3-5 ml) and the mucosa is treated with the cotton ball. A 2 or 3 per cent solution is sometimes used for the purpose. Overdosage can cause severe toxic effects. Lethal outcomes were reported from incorrect use of dicaine.

The preparation is available in powder form. It should be stored in well stoppered containers.

*Lidocaine hydrochloride* (xylocaine) is a yellowish white crystalline powder with bitter taste, soluble in water and alcohol. It is a strong local anaesthetic. Its anaesthetic effect is stronger and longer than that of procaine, while toxicity is comparatively low. Its 0.25-0.5 per cent solutions are used for infiltration anaesthesia, while 0.5-1-2 per cent solutions for conduction anaesthesia. Lidocaine is used for endoscopic manipulations (irrigation or treatment of mucosa with medicinal agents), in arrhythmias, especially in ventricular extrasystole, during operations on the heart, and for angiography. When given in therapeutic doses, lidocaine has a mild vasoplegic and hypotensive effect. The preparation is injected intravenously as a 2 per cent solution (2.5-5 ml) in 5 per cent glucose. The dose should

be injected slowly, within 1-2 minutes. The anaesthetic effect lasts for 15-20 minutes. The control of ECG is necessary. The preparation is contraindicated in cases with cardiovascular insufficiency, atrio-ventricular block (II and III degree), and in hepatic and renal dysfunction.

The preparation is available in 2-ml ampoules (2 per cent solution). It should be kept in the dark.

*Cinchocaine hydrochloride* (sovcaine) is a white crystalline powder readily soluble in water and alcohol. It is 15-20 times more active than procaine, and 15-20 times more toxic. Its withdrawal from the body is slow. The preparation is mainly used for spinal anaesthesia in a dose of 0.5-0.7 ml of a 0.5-1 per cent solution (with all precautions). The preparation can reduce arterial pressure (1 ml of a 5 per cent ephedrine solution should first be injected subcutaneously).

The preparation is available in 1-ml ampoules containing 0.5 and 1 per cent solutions. The preparation is also produced in powder form. It should be kept in tightly stoppered bottles of orange glass.

### ANALGESICS

*Morphine hydrochloride* has a powerful analgesic and sedative effect, it causes sleepiness and euphoria, inhibits respiration, slows down the heart rate, causes vasoplegia, stimulates the vomiting centre, inhibits the gastrointestinal secretion, and increases the tone of smooth muscles of the bronchi and the bladder. Morphine preparations are called narcotic analgesics. Morphine is used for premedication. It is given subcutaneously, 40-50 minutes before an operation. Its effect is attained in 10-15 minutes and lasts for 3-5 hours. The preparation is used for anaesthesia with deep central analgesia. Morphine preparations are used postoperatively to alleviate pain and as a sedative. In order to remove side-effects (nausea, vomiting, bradypnoea), it is recommended that morphine preparations are given with cholinolytic preparations.

The preparation is produced in powder form and as a 1 per cent solution (in 1-ml ampoules). Morphine preparations should be kept in the dark (under lock).

*Omnopon* (pantopon, domopon) is similar in its action to morphine and it is used for the same indications. It is less popular than morphine or promedol though.

The preparation is produced as powder or as 1 and 2 per cent solutions (in 1-ml ampoules). It should be kept in a dark and cool place (under lock).

*Trimeperidine hydrochloride* (promedol) is similar to morphine in its effect on the central nervous system but its analgesic effect is somewhat weaker, the toxicity is markedly lower, its inhibiting effect on the respiratory system is lower and the action on the blood

circulation is milder, it provokes vomiting less frequently. The preparation is used as an analgesic in various diseases and injuries. It is widely used for premedication for surgery, during postoperative period, and also for general anaesthesia to strengthen the analgesic effect. The effect lasts for 3-4 hours. The preparation is well tolerated by the patients. Among aftereffects are nausea, dizziness, and weakness.

The preparation is produced as powder, in 0.025 g tablets, or as 1 and 2 per cent solutions in 1-ml ampoules. It should be kept under lock.

*Phentanyl* (sentonyl, fentanest) is a rapid-acting agent with a strong but short-acting analgesic effect. Its analgesic activity is 100 times higher than that of morphine. The maximum effect is attained in 1-2 minutes after its intravenous injection and in 10-20 minutes after intramuscular or subcutaneous administration. The analgesic effect lasts not longer than 30 minutes. The preparation has an inhibiting effect on the respiratory function and can cause apnoea and marked bradycardia relieved by atropine. The preparation is widely used for neuroleptanalgesia in combination with neuroleptics.

Droperidol and phentanyl are manufactured as a ready-mixed preparation thalamonal. The preparation is available in the form of 0.005 per cent solution in 2-ml ampoules and 10-ml vials. It should be kept under lock.

*Dipidolor* (piritramid) has a pronounced analgesic effect (twice as effective as morphine), causing no vomiting or respiratory distress. It can be used post-operatively to relieve pain, in patients showing habituation for morphine and promedol. The analgesic effect lasts for 3-4 hours, if the dose is doubled, the effect lasts for 10-14 hours.

The preparation is produced in 2-ml ampoules containing 15 mg of the preparation.

*Pentazocine* (levir, fortal) does not differ substantially from morphine in its analgesic activity. Nausea, vomiting, intestinal paresis, retention of the urine, or respiratory distress occur less frequently and are less pronounced. The anaesthetic effect lasts for 3-4 hours. The preparation does not affect blood circulation. It has good local and general tolerability. Contraindications for its use are renal and hepatic failure.

The preparation is produced in 1-ml ampoules containing 30 mg of pentazocine. The preparation should be kept under lock.

#### NEUROLEPTICS, SEDATIVES, AND TRANQUILIZERS

*Aminazine* (megaphen, plegomazin) has a marked sedative effect. Large doses can cause a condition close to physiological sleep. It potentiates the effect of anaesthetics, hypnotics, analgesics, and

**local anaesthetics** The preparation is an effective antiemetic. It effectively relieves hiccup and is a strong adrenolytic preparation. It causes tachycardia, hypotension, and peripheral vasoplegia. The preparation is mainly used as an antiemetic and a peripheral spasmolytic. The preparation can also be used for premedication. It inhibits thermoregulatory mechanisms and is used for hyperpyrexia.

The preparation is available in 0.025, 0.05 and 0.1 g coated tablets, and as a 2 per cent solution in 1, 2, and 5-ml ampoules. The preparation should be kept in the dark.

*Propazine's* (amprazine, frenyl) action is similar to that of aminazine. Its sedative effect is slightly inferior while the antihistaminic action is more pronounced. Its toxicity is lower. Indications and contraindications for the use of propazine are the same as for aminazine.

The preparation is produced in 0.025 g coated tablets and as a 2.5 per cent solution in 2-ml ampoules. The preparation should be kept in the dark.

*Haloperidol* (aloperidin, haldol) has a pronounced neuroleptic and tranquilizing action. It potentiates the effect of hypnotics, anaesthetics, and analgesics. Its adrenolytic effect is low. The preparation is administered together with analgesics, hypnotics and other neurotropic preparations during preoperative medication. The preparation is prescribed for peroral, intravenous and intramuscular administration. Extrapyramidal disorders associated with the use of haloperidol can be relieved by aminazine or atropine. Organic affections of the kidneys are contraindications for the use of haloperidol. The preparation is produced in the form of 0.0015 and 0.005 g tablets, as a 0.5 per cent solution in 1-ml ampoules, and 0.2 per cent solution in 10-ml vials (ten drops of the solution are equivalent to 1 mg of haloperidol).

*Droperidol* (droleptan, inapsin) is a strong rapid- and short-acting neuroleptic preparation. It is also an efficient antishock and anti-emetic preparation. When administered intravenously, the effect is observed in 2-3 minutes attaining its maximum in 10-15 minutes and lasting for 30-40 minutes. The action decreases gradually within 2-4 hours. Practically there are no side-effects. Its neuroleptic effect is three times stronger than that of aminazine. The preparation is used for premedication and anaesthesia in combination with the analgesic phentanyl (neuroleptanalgesia). The preparation is contraindicated for extrapyramidal disorders. Care should be exerted in giving the preparation to patients treated with hypotensive drugs since the arterial pressure may suddenly fall after the administration of droperidol.

The preparation is available in 10-ml vials containing 25 mg. The preparation should be stored with precautions.

*Thalamonal* is a complex preparation containing 2.5 mg of droperidol and 0.05 mg of phentanyll in 1 ml. The preparation is used for anaesthesia, for elimination of lung oedema and attacks of renal and hepatic colics, in traumatic shock, and for premedication of patients before various diagnostic and therapeutic manipulations. The preparation is injected intramuscularly or intravenously (1-5 ml).

Thalamonal is produced in 10-ml vials. The preparation should be stored with precautions.

*Chlordiazepoxide* (elenium, napoton) is a hypnotic preparation which acts sedatively on the central nervous system, it also has anticonvulsive effect and causes relaxation of muscles. The preparation is given in 2.5-5 mg doses two times a day for premedication and during the postoperative period. The preparation is contraindicated for acute diseases of the kidneys, liver, and in severe myasthenia. It should not be used with inhibitors of monoaminoxidase or phenothiazine derivatives.

The preparation is produced in 0.01 g coated tablets, should be stored with precautions.

*Diazepam* (seduxen, relanium) is similar to chlordiazepoxide but is more effective in smaller doses. It has minimum effect on the cardiovascular system and a pronounced sedative and hypnotic effect, it relaxes muscles and has anticonvulsive action. The preparation is used for premedication in 5-10 mg doses which are given 60-90 minutes before the operation. It is effective for cramps of various genesis, given in 2.5 or 10-20 mg doses, which are injected intravenously slowly in 20 ml of a 40 per cent glucose solution. If the preparation is injected rapidly, it may cause hypotension and respiratory distress. The preparation is contraindicated for acute diseases of the liver, kidneys, and severe myasthenia.

Diazepam is produced in 0.005 g tablets and 0.5 per cent solution in 2-ml ampoules. The preparation should be stored with precautions.

*Trioxazine* (trimetozine, sedoxazine) is a tranquilizer. It potentiates the effect of narcotic and hypnotic agents. The preparation is used for premedication of patients. Big doses of trioxazine can cause nausea, weakness, sleepiness, and dry mouth mucosa. The preparation is produced in 0.3 g tablets that should be stored with precautions.

## MUSCLE RELAXANTS

Muscle relaxants are substances that block selectively the reflex arch in the neuromuscular synapse. Owing to the specific selectivity of the preparations, they relax the muscles without inhibiting the central nervous system or other vital body functions. Muscle relaxants are most effective during operative anaesthesia because it becomes possible to carry out an operation with shallow anaesthesia with low and harmless anaesthetic concentrations in the blood.

*Mechanism of action* Muscle relaxants act on the neuromuscular synapse. It seems necessary, before describing the mechanism of action of muscle relaxants, to discuss in short the modern concepts of the neuromuscular synapse and processes occurring in it. It is believed now that the transition of excitation from the nerve onto the skeletal muscle is effected with involvement of the chemical agent known as mediator acetylcholine. But the chemical mechanism of conduction of excitation obligatory includes the elements of electrophysiological factors. As a nervous fibre approaches the muscle, it ramifies and each branch leads to a separate muscle spindle. Here the nerve cell (neuron) loses its myelin sheath and as it fuses with the cells of the muscle fibre, thus forming a neuromuscular synapse. The interaction between the nerve and the muscle in this region is effected via membranes. The exposed portion of the nerve which opposes the surface of the muscular fibre is coated with a terminal membrane, while the exposed portion of the muscular fibre ends with a terminal plate. The side of the plate in apposition to the terminal membrane of the nerve cell is called postsynaptic. There is a microscopic slit between the postsynaptic membrane and the terminal membrane. This is called subneural space. A system of acetylcholine (including various states of this mediator connected with the protein complexes and other substances, such as choline-acetylase, acetylcholinesterase, choline receptor and bound acetylcholine) is found in the region of the neuromuscular synapse, inside the cell. When an impulse passes along the nerve fibre and reaches the neuromuscular synapse, acetylcholine is released from its protein complex and passed through the synaptic slit to interact (by adsorption) with the protein receptor found on the postsynaptic membrane. Adsorption of acetylcholine alters permeability of the postsynaptic membrane to various ions. Permeability to the potassium ion, and especially sodium ion, increases. Positively charged potassium ions and negatively charged chloride ions are accumulated on the inner surface of the postsynaptic membrane, while the positive sodium ion is accumulated on the outer surface. Uneven diffusion of the ions polarizes and then depolarizes the membranes. This accounts for excitation and inhibition processes.

A resting postsynaptic membrane is polarized, i.e. its inner surface is electronegative with respect to the outer surface. The electrostatic difference between the inner and outer surfaces is called the resting potential (static polarization). Changes in permeability of the postsynaptic membrane caused by the action of acetylcholine and by the passage of a great quantity of the sodium ions to the inner surface of the membrane decrease its electronegativity. Thus, the potential difference between the surfaces of the postsynaptic membrane decreases, i.e. depolarization of the terminal plate occurs. The action potential arises. As soon as the action potential attains a certain magni-

tude, it acts as a stimulant on the neighbouring portion of the muscle fibre to cause its depolarization and contraction. The terminal plate remains depolarized for 2-3 ms because acetylcholine is quickly destroyed by acetylcholinesterase. The cholinergic receptor is released to accept another portion of acetylcholine. Acetylcholine is resynthesized under the action of choline acetylase and is bound again with the protein complexes. As soon as the cholinergic receptor of the terminal plate becomes free from acetylcholine, the selective permeability of the postsynaptic membrane is regained. The potassium and sodium ions diffuse to their place, the terminal plate is repolarized, and the resting potential is restored. The transmission of excitation from the nerve to the muscle and its contraction are only possible on condition that a process of transition from static polarization to depolarization (and the reverse) is constantly maintained in the neuromuscular synapse and the muscular fibre. Disturbance in any link of this continuous process (static polarization-depolarization-repolarization) disturbs the muscular activity.

The effect of most relaxants on the neuromuscular synapse is based on competition of these substances with acetylcholine for the receptors of the terminal plate. Muscle relaxants take up the receptors sooner than acetylcholine and block the action of the mediator.

*Classification* All muscle relaxants can be divided into several groups depending on their properties, such as the chemical structure, time of action, effect on the patient, and the mechanism of their action. By the mechanism of action muscle relaxants can be divided into two substantially different groups: peripheral relaxants (mainly the relaxants used in clinical conditions) and central relaxants which block synapses of the spinal cord (mephenesine and glyceroguaiacol ester). Peripheral relaxants are divided into the following three groups:

- 1 Non-depolarizing relaxants—the preparations preventing reaction between acetylcholine and the receptor, thus inhibiting depolarization. They have no practical effect on the physical, chemical or any other properties of the receptors of the terminal plate nor does acetylcholine cause depolarization of the terminal plate. This group includes *d*-tubocurarine (tubarine), flaxedil, dypacin, and some others.

- 2 Depolarizing relaxants—the preparations causing stable depolarization of the terminal plate after the first, second, and sometimes the third administration, and inhibiting repolarization. After adsorption on the receptors they act like acetylcholine and cause depolarization. Unlike acetylcholine (which is decomposed in thousandth fractions of a second) they ensure depolarization that can last for a few minutes, during which repolarization of the postsynaptic membrane of the terminal plate is impossible. After repeated

administrations these relaxants cause antidepolarization block. The group includes succinylcholine chloride and decamethonium

3 Mixed relaxants are preparations causing depolarization and then non-depolarization block after a single injection in all cases

### Non-depolarizing Muscle Relaxants

*Tubocurarine chloride* (tubarine) blocks mainly *n*-choline reactive systems of the skeletal muscles and has a strong myoparalytic effect. Its action (after an intravenous administration) develops gradually and the muscles are relaxed in 1-2 minutes. Myoplegia begins with the muscles of eyes and eyelids, then it passes onto the muscles of the extremities and the abdomen. The diaphragm relaxes the last. Complete relaxation lasts for 20-25 minutes. Repeated doses should be  $1\frac{1}{2}$ -2 times smaller. The preparation has no substantial effect on the vital body functions. It decreases slightly the arterial pressure and can sometimes cause bronchial spasm. The antagonists to tubocurarine are proserin and galanthamine, which are usually administered with atropine.

The preparation is available in the form of a 1 per cent solution in 2- and 5-ml ampoules. The preparation should be kept under lock.

*Diplacine* is similar to tubocurarine by its action. The maximum effect is attained in 5-6 minutes following the intravenous injection and lasts for 30-40 minutes. Since the preparation has marked cumulative effect, a repeated dose should be decreased 2 or 3 times. The preparation is not used widely because of the high variation in the time of its action.

The preparation is produced as a 2 per cent solution in 5-ml ampoules. The preparation should be kept in the dark.

*Dioxonium* is a muscle relaxant of the mixed action. It first causes depolarization and then non-depolarization. Following an intravenous administration, the muscles are relaxed in  $1\frac{1}{2}$ -2 minutes and remain so for 20-40 minutes. Whenever necessary,  $\frac{1}{2}$  or  $\frac{1}{3}$  dose is given additionally. The antagonists to the preparation are proserin and galanthamine.

*Dioxonium* is produced in the form of a 0.1 per cent solution in 0.5-ml ampoules. The preparation should be kept in the dark.

*Pavulon* is a neuromuscular relaxant. It is a rapid-acting preparation. It does not cause bronchospasm or affect arterial pressure. The preparation is used during operations on the abdominal and thoracic organs, in ophthalmology and orthopaedics. The maximum effect is attained in  $2-2\frac{1}{2}$  minutes and the action continues for about 45 minutes. The preparation is injected intravenously. Contraindications to this preparation are myasthenia and diseases of the kidneys. The preparation is produced in 2-ml ampoules (1 ml of the solution contains 2 mg of the preparation).



*Diadonium* is a short-acting non-depolarizing relaxant. The muscles are relaxed in 15-40 seconds and remain so for 5-10 minutes. When administered repeatedly, the dose should be decreased  $1\frac{1}{2}$  times. The preparation causes dilation of the pupils which remain widened for 25-30 minutes. The antagonists to diadonium are proserin and galanthamine.

The preparation is produced in powder form and as a 2 per cent solution in 2-ml ampoules.

### Depolarizing Muscle Relaxants

*Dithylin* (succinylcholine chloride, myo-relaxin) causes twitching of the muscles of the face, neck and extremities in 10-15 seconds after administration, twitching continues for 15-20 seconds and the muscles then become relaxed completely with apnoea for 5-7 minutes. Complications are usually absent except bradycardia and increased intraocular pressure in some cases. Dithylin can be used during intubation of the trachea, diagnostic manipulations, and minor operations.

The preparation is available as a 1 per cent solution in 2-ml ampoules and vials containing 0.1, 0.25 and 0.5 g of the preparation. The preparations should be kept under lock.

## CARDIOVASCULAR PREPARATIONS

### Cardiac Glycosides

*Digoxin* (digolan, lanicoi), as other preparations of foxglove, act on the heart muscle. It slows down markedly the heart rate and has a strong diuretic effect. The preparation is used to treat acute heart failure and weakness during operations, in the postoperative period, and also in arrhythmias. The preparation is administered intravenously in 5, 20 or 40 per cent glucose solutions.

The preparation is available in the form of tablets, and 0.025 per cent solution in 2-ml ampoules. Digoxin should be stored in the dark.

*Strophanthin K* is only slightly active when taken per os but its intravenous administration gives a rapid and strong effect by intensifying ventricular systole. It has only slight effect on the heart rate or conduction of the atrioventricular node. The preparation is used for acute heart failure and paroxysmal tachycardia. Strophanthin is injected slowly in a 5, 20 or 40 per cent glucose solution. Contraindications to the use of strophanthin are cardiac arrhythmia, organic heart and vessel defects.

The preparation is available in the form of a 0.05 per cent solution in 1-ml ampoules. The preparation should be kept in the dark.

*Corglycon* has a milder though longer effect than *striophanthin*. The indications for its use are the same as those for *striophanthin* and *digitalis*. The preparation is intended for slow intravenous injection.

The preparation is available in 0.06 per cent solution in 1-ml ampoules. It should be kept in the dark.

### Anti-arrhythmic Preparations

*Novocainamide* (amidoprocaine, procainamide) decreases excitability and conduction of the heart muscle and inhibits the activity of ectopic foci of automatism. It prevents and treats arrhythmias. The preparation is intended for slow intravenous injections. The administration may be followed by general weakness, headache, nausea, vomiting, excitation, insomnia, and bitter taste in the mouth. Contraindications: heart block.

*Novocainamide* is available in 0.25 g tablets and as a 10 per cent solution in 5-ml ampoules. The preparation should be kept in the dark.

*Anaprilin* (nifedipine, nifedipine) weakens the effect of the sympathetic impulsion on the  $\beta$ -adrenergic blockers, slows down the heart rate, decreases myocardial contractility, and the cardiac output. *Anaprilin* is used as an antiarrhythmia preparation. It is intended for slow intravenous injection with ECG control. Bradycardia, hypotension, nausea, weakness, insomnia, and diarrhoea are sometimes observed. Contraindications to the use of the preparation are disorders in the atrioventricular conduction, heart block, and tendency to bronchospasm.

The preparation is produced in 40 mg tablets and in 5-ml ampoules containing 5 mg of the preparation. The preparation should be kept in the dark.

*Verapamil* (isoptin, diltiazem) dilates the coronary vessels and decreases the arterial tone. It is especially effective in treating paroxysmal supraventricular tachycardia, threatening types of fibrillation tachycardia, and ventricular extrasystole. Contraindications: cardiac shock and atrioventricular heart block. The preparation should be used with precautions in bronchial asthma.

*Verapamil* is produced in 40 mg coated tablets and 2-ml (5 mg) ampoules.

### Spasmolytic, Vasodilating, and Hypotensive Preparations

*Papaverine hydrochloride* decreases the tone and relaxes smooth muscles, and has a mild sedative effect. It eliminates spasms of peripheral vessels, prevents attacks of bronchial asthma and bronchospasm. The preparation is injected intravenously and subcutaneously.

Papaverine is available in the form of 0.02 g tablets, and 2 per cent solution in 2-ml ampoules

*Aminophylline* (aminocardol, diaphylline) has a vasodilating and spasmolytic effect, it dilates the coronary vessels, relaxes the bronchial muscles, and intensifies the renal blood flow. It is effective when given together with cardiac glycosides in emergency therapy and regulates the water-electrolyte metabolism during operations and in the post-operative period. The preparation is intended for slow intravenous injections. Contraindications: pronounced hypotension and arrhythmia.

Aminophylline is produced in powder and tablets of 0.05 g, as 0.2 g suppositories, and as a 24 per cent solution (in 2-ml ampoules) and a 2.4 per cent solution (in 10-ml ampoules). The preparation should be kept in the dark.

*Dibazol* (tromasedan, bendazole) has a vasodilating, spasmolytic and hypotensive effect, it also stimulates the function of the spinal cord. The preparation is used during anaesthesia and post-operative period. It is intended for intravenous and subcutaneous injections. The preparation is produced in 0.02 g tablets, as powder, as a 1 per cent solution (in 1-ml ampoules), and a 0.5 per cent solution (in 2-ml ampoules).

## OTHER PREPARATIONS USED FOR ANAESTHESIA, RESUSCITATION, AND INTENSIVE THERAPY

### Hypnotics

*Barbital* (barbitone, barbitural) is a white crystalline powder without odour, it is soluble in boiling water and alcohol. Its action is durable. The sleep is induced in 30-40 minutes and lasts to ten hours. The preparation is destroyed in the body rather slowly, and is mainly withdrawn from the body in the unchanged form by the kidneys during 5-11 days. If the renal function is upset, the preparation is accumulated in the body.

Barbital is used for premedication, as a sedative and hypnotic means, especially in patients who readily fall asleep but wake at night. A single dose is from 0.01 to 0.25 g to children aged under 7, and 0.3 g to older children.

The preparation is available in the form of powder and 0.25 g tablets. Barbital should be stored with precautions.

*Barbital sodium* (medinal) is a white crystalline odourless substance soluble in water. It has a prolonged hypnotic effect. Unlike barbital, it is more rapid-acting and less toxic. The indications are the same as for barbital. The preparation is given before night sleep on the eve of operation.

Barbital sodium is available in 0.3 g tablets and powder. It should be stored with precautions.

*Phenobarbital* (luminal, sedonal) is a white crystalline powder without odour with a bitter taste, soluble in water and alcohol. It is a long-acting hypnotic and anticonvulsant preparation. It is given before night sleep together with antihistaminic preparations.

The preparation is produced as powder and tablets of 0.05 and 0.1 g. It should be stored with precautions.

*Barbamyl* (amytal sodium) is a white fine crystalline powder without odour, readily soluble in water. The patient sleeps for about 6-8 hours. It is quickly decomposed in the liver, is not accumulated in the body, except in patients with diseased liver. The sedative effect of barbamyl is intensified if antihistaminic preparations e.g. suprastin, dimedrol, pipolphen, are given simultaneously. The preparation is given per os before night sleep on the eve of operation.

The preparation is produced as powder and 0.1 and 0.2 g tablets that should be stored with precautions.

*Chloral hydrate* (aquachloral, chloradorm) is a sedative, hypnotic and analgesic preparation. When given in big doses, it produces a narcotic effect. It is also used as an anticonvulsant (by enema). To that end, 1 g of the preparation is mixed with 25 ml of a viscous substance and 25 ml of distilled water. The sleep is induced in 10-20 minutes and the patient sleeps for 5-6 hours. Hypotension is frequently observed.

The preparation is produced in powder and 0.5, 0.7 and 1.5 g tablets. Chloral hydrate should be kept in tight containers in the dark.

### Antihistaminic Preparations

*Dimedrol* (alledryl, alleigan) lessens the reaction to histamine. It also has a sedative effect and intensifies the action of hypnotics and analgesics; it has a mild antiemetic property. The preparation can be used to lessen the body response during transfusion of blood or its substitutes; its 10 per cent solution is used for local anaesthesia during rhinological operations. The preparation is also given per os, intramuscularly, intravenously, and locally.

The preparation is produced in powder form, in 0.05 g tablets, and also as a 1 per cent solution in 1-ml ampoules. The preparation should be stored with precautions.

*Diprazin* (pipolphen, antiallersin). In addition to antihistaminic properties, the preparation influences the central nervous system, potentiates the hypnotic, narcotic, and analgesic effects of other preparations, it is effective against hiccup and has a pronounced antiemetic property (decreases excitability of the vomiting centre). It also has a vasodilating effect, increases sensitivity to blood loss,

and gives a moderate peripheral cholinolytic effect, it produces hypothermic action explained by intensified heat withdrawal due to vasoplegia with predominant dilation of the skin vessels and decreasing motor activity. The preparation is destroyed mainly by the liver where it is inactivated by 70-90 per cent. Dipiazin is well tolerated by the patient. Nausea, dryness and moderate anaesthesia of the mouth mucosa sometimes occur. Infiltrations are possible in intramuscular injections.

The preparation is produced in 0.025 g coated tablets, which should be stored with precautions.

*Suprastin* (chloropyramine, synopen) has a marked antihistaminic effect, it also has sedative properties. The preparation is used for premedication and for various allergic reactions. The preparation is administered per os, intramuscularly or intravenously.

The preparation is produced in 0.025 g tablets and as a 2 per cent solution in 1-ml ampoules. Should be kept under lock.

### Anticholinesterase Preparations

*Proserine* (neostigmine methylsulfate) has pronounced anticholinesterase activity. It is an antagonist to the curare-like preparations. It blocks cholinesterase and stimulates accumulation of acetylcholine with subsequent displacement of the curare-like substances from cholinergic receptors. The administration of proserine causes bradycardia and hypersalivation, atropine (0.3-0.5 mg) should therefore be given preliminarily. Contraindications: epilepsy, hyperkinesia and bronchial asthma. The antagonists to proserine are atropine and methacin.

Neostigmine methylsulfate is produced as a 0.05 per cent solution in 1-ml ampoules. The preparations should be kept in the dark.

*Galanthamine* is similar to proserine by its action. Being a strong inhibitor of cholinesterase, it increases the body sensitivity to acetylcholine. The preparation is administered intravenously. Contraindications: bradycardia, angina pectoris, hyperkinesia, and bronchial asthma.

The preparation is produced as a 0.25, 0.5 and 1 per cent solution in 1-ml ampoules. Galanthamine should be kept under lock.

### Cholinolytic Preparations

*Atropine sulphate* blocks *m*-choline-reactive systems of the body to make them insensitive to acetylcholine. It lessens secretion of the salivary and sweat glands, decreases the tone of smooth muscles, dilates the pupils, and increases the intraocular pressure. The preparation is intended for intramuscular or subcutaneous injections, 30-40 minutes before the anaesthetic effect is desired, or directly in

the operating room, 2-3 minutes before the operation (intravenously) to ensure reliable vagolytic effect

The preparation is produced as powder or 0.1 per cent solution (in 1-ml ampoules)

*Scopolamine hydrobromide* is similar in structure to atropine and its action on the human organism is similar as well. Unlike atropine, scopolamine has sedative properties and causes sleepiness and amnesia. Like atropine, it is used for premedication together with analgesics and hypnotics. The preparation is produced as powder and a 0.05 per cent solution in 1-ml ampoules.

*Methacin* has a selective peripheral *m*-cholinolytic action. It is twice as active as atropine, decreases the secretion of the mucosa, and removes spasms when injected intravenously. It has a similar effect on the cardiovascular system, but its action on the dilation of the pupil is ten times less effective. The routes of administration are the same as for atropine.

The preparation is produced as a 0.1 per cent solution in 1-ml ampoules and should be stored in the dark.

### Ganglioblocking Preparations

*Benzohexonium* (hexonium) is used for controlled hypotension during operation, to prevent vegetative reflexes, and in lung oedema. The preparation is administered intravenously, intramuscularly and subcutaneously. About 50 per cent of the preparation are withdrawn from the body by the kidneys in the unchanged form and the remaining preparation is excreted as metabolites within 24 hours. The preparation may cause tachycardia, dryness in the mouth, and decrease the tone of the intestine. Contraindications: shock, hypotension, affections of the kidneys and the liver, degenerative changes in the central nervous system, disorders in the blood coagulating and anticoagulating systems.

The preparation is produced in 0.1 g tablets and as a 2.5 per cent solution in 1-ml ampoules. The preparations should be stored in tightly stoppered containers.

*Arfonad* (trimethaphan camphorsulfonate) is a strong short-acting ganglioblocking agent. It acts directly on the smooth muscles of the vessels and promotes the release of histamine. It causes a mild hypotension in the veins, venous haemorrhage is therefore possible. The preparation is used for controlled hypotension, in lung oedema, and during operations on the heart with extracorporeal blood circulation and vessels. The preparation is administered by intravenous drip because intramuscular and subcutaneous injections cause stable and almost uncontrollable hypotension. The preparation acts in 2-4 minutes after the administration, in 4-6 minutes after discontinuation of administration the arterial pressure rises to attain its initial level in

15-20 minutes Contraindications anaemia, acute blood loss, severe diseases of the liver, kidneys, heart, and the central nervous system  
 Arfonad is produced in powder form in 250-mg vials

### Adrenaline and Adrenomimetic Preparations

*Adrenaline* (epinephrine) acts similar to the effect of excitation of the sympathetic nerves. It accelerates the heart rate and intensifies the heart contractions to increase the stroke volume. Adrenaline contracts the vessels of the kidneys, skin and subcutaneous cellular tissue, contraction of the pulmonary vessels and the vessels of the skeletal muscles is less pronounced. Adrenaline dilates the vessels of the heart and brain, and increases the arterial pressure. Adrenaline causes relaxation of the muscles in the bronchi and the intestine, intensifies the tissue metabolism, and increases the glucose level in the blood. The preparation is administered subcutaneously, intramuscularly, and intravenously. It is used for cardiac weakness to eliminate attacks of bronchial asthma, and for allergic reactions. Adrenaline is administered directly into the heart in case of its arrest.

Adrenaline is produced as a 0.1 per cent solution in 1-ml ampoules. The preparation should be kept in the dark.

*Noradrenaline hydrotartrate* (arterenol, levarterenol). As distinct from adrenaline (epinephrine), it has a less marked effect on the heart and a strong vasoconstrictor effect. It has a mild broncholytic action and is a mild stimulant of metabolism. Noradrenaline is used for arterial hypotension caused by decreased tone of the peripheral vessels (collapse), it is also administered for stabilization of arterial pressure during operations on the sympathetic nervous system. Noradrenaline hydrotartrate is administered by intravenous drip only.

The preparation is produced as a 0.2 per cent solution in 1-ml ampoules. It should be kept in the dark.

*Ephedrine hydrochloride* (ephedrosan, neo-fedrin) has the same pharmacological action as adrenaline but it is a short-acting agent. It increases excitation of the respiratory centre, stimulates the central nervous system, and is used for allergic conditions, hypotension during operations, blood loss, and injuries. Its effect is marked in 30-60 seconds after intravenous administration, the action lasts for 10-20 minutes.

Ephedrine hydrochloride is produced as powder, in 0.025 g tablets, and as a 5 per cent solution in 1-ml ampoules. The preparation should be stored in the dark.

*Antasthmín* (isadrin, euspiran) is similar to adrenaline and noradrenaline by its action. It has a strong broncholytic effect. The vasoconstrictor action is much weaker than that of adrenaline. The

toxicity of the preparation is low. The preparation is used during operations on the heart and vessels and for treating bronchial asthma. It is administered by intravenous drip. Possible side-effects are palpitation, dry throat, and a slight elevation of the arterial pressure.

The preparation is produced as a 0.05 per cent solution in 1-ml ampoules and a 1 per cent solution in 100-ml vials. The solution should be kept in the dark.

### Anti-adrenergic Preparations

*Phentolamine* (regitine, dibasin) is an  $\alpha$ -adrenoblocking agent. It relieves spasms of arterioles and precapillaries and improves the peripheral circulation of blood. The preparation is used during operations for pheochromocytoma and in conditions attended by hyperadrenalinaemia. Phentolamine is intended for intravenous administration in fractional doses in order to prevent a sudden fall of the arterial pressure. Contraindications: marked cardiovascular failure.

Phentolamine is produced as a 0.5 per cent solution in 1-ml ampoules. The preparation should be kept in the dark.

### Diuretics and Dehydrating Agents

*Urea* (carbamide, ureaphil) increases diuresis by increasing osmotic pressure in the tubules, and decreases intracranial pressure. The preparation is used to prevent and treat brain oedema, especially at its early stages. A 30 per cent solution in 10 per cent glucose is used for the purpose. The effect is attained in 60-90 minutes and lasts for 5-6 hours. Contraindications: hepatorenal failure, marked cardiovascular failure, and suspected intracranial haemorrhage.

Urea is produced as a dry substance in 30, 45, 60, and 90-g vials. To each vial of urea a diluent (10 per cent glucose solution in a vial) is attached.

*Furosemide* (lasix, furanthril) is a strong diuretic. Its diuretic effect is explained by the depressed reabsorption of the sodium and chloride ions in the renal tubules. The preparation is especially efficacious for alkalosis and acidosis. It is effective in a few minutes after administration and the action lasts for 1.5-3 hours. The preparation is used for oedema of the lungs and brain and in other emergency conditions. Contraindications: acute glomerulonephritis, liver cirrhosis, hypokalaemia, and overdosage of digitalis preparations. Potassium salts should be given to the patient during treatment with furosemide.

The preparation is produced as a 1 per cent solution in 2-ml ampoules and as 0.04 g tablets.



*Mannite* (mannitol, osmosal) in a hypertonic solution acts as a strong diuretic by increasing the osmotic pressure of the plasma and by decreasing water reabsorption. Mannitol is also used to decrease intracranial pressure or oedema of the brain, and for acute hepatorenal failure. The preparation is also used to prevent ischaemia of the kidneys during operations with extracorporeal blood circulation. The preparation is injected intravenously in an isotonic sodium chloride solution. Contraindications: renal excretory dysfunction.

Mannitol is produced in 500-ml vials. The preparation should be stored at temperatures not above 20°C.

*Spironolactone* (aldactone, verospiron) is an antagonist to aldosterone, it prevents sodium reabsorption in the kidneys and decreases excretion of potassium and urea. The preparation is used during the post-operative period in patients with cardiac decompensation, in ascites and oedemas of other aetiology. Among side-effects are adynamia, sleepiness, and skin eruptions. The preparation is contraindicated for acute renal failure.

The preparation is produced as 0.025 g tablets.

### Preparations Acting on Blood Coagulation

*Heparin* (pularin, vetren) is an anticoagulating agent of direct action. It has a direct effect on the native blood coagulating factors. It also inhibits the formation of thrombin and prevents agglutination of thrombocytes. Heparin is used to prevent and treat thromboembolic complications, sepsis, and during resuscitation. Coagulability of blood should be controlled during administration of heparin.

The preparation is available in 5-ml vials (1 ml contains 5000 units of the preparation).

*ε-Aminocaproic acid* (amicar) inhibits the fibrinolytic activity, decreases the activity of plasminogen and partly of plasmin, it has a haemostatic effect in haemorrhages connected with increased fibrinogen content. The preparation is used to prevent and arrest haemorrhages in surgical operations and various pathological conditions associated with increased fibrinolytic activity of blood and tissues.

The preparation is available in 100-ml vials of sterile solution.

*Fibrinogen* is used for conditions connected with markedly decreased content of this blood enzyme (profuse bleeding, blood transfusions after operations on the heart, in haemophilia A). The preparation is administered by intravenous drip. Contraindications: phlebitis and thrombosis.

Fibrinogen is available in 250-ml and 500-ml bottles containing 0.9-1 and 1.8-2 g of fibrinogen, respectively.

## Hormones and Their Analogues

*Cortisone acetate* (adreson, cortadien) is mainly used for acute or chronic functional insufficiency of the adrenal cortex in surgical patients in critical conditions after operations, hypoxia, injuries, fever, peritonitis, etc. The preparation can be administered straight during operation. Hydrocortisone or prednisolone should however be preferred. If the post-operative course is normal, the preparation should be given for 4-5 days, in case of complications, the course should be prolonged for 6-10 days.

Cortisone is produced in the form of 0.025 g tablets and suspension in 10-ml vials (1 ml contains 0.025 g of the preparation). Cortisone should be stored in the dark.

*Hydrocortisone acetate* (abbocort, hydrison) is similar to cortisone by its action but it is more active. The preparation is used for acute cardiac failure, shock, blood loss, acute vascular collapse, burns, and in disorders of the blood coagulating system.

The preparation is available in 5-ml ampoules (125 mg) for intramuscular injections.

*Prednisolone* (codelcortone, prenolone) is a dehydrated analogue to hydrocortisone and similar to it by its pharmacological properties. The preparation has the same uses. It is intended for intravenous or intramuscular administration.

The preparation is available in 0.005 g tablets and as a 3 per cent solution in 1-ml ampoules.

*Dexamethasone* (decardon, decacortin) is an analogue to other glucocorticoids but having a higher activity (about 7 times higher than prednisolone and 35 times higher than cortisone). It has a more pronounced anti-inflammatory and anti-allergic action. The preparation is well tolerated by the patients, therapeutic doses have no effect on the electrolyte metabolism, nor does the preparation promote retention of sodium or water in the body. Dexamethasone has the same uses as hydrocortisone. The preparation is mainly administered by intravenous drip or in fractional doses.

Dexamethasone is produced as 0.5 mg tablets and in 1-ml ampoules (4 mg of the preparation).

*Retabolil* (deca-durabolin, hormoretard) has a favourable effect on the nitrogenous metabolism, retaining nitrogen in the body and decreasing excretion of urea by the kidneys. It also causes retention of sulphur, potassium and phosphorus that are necessary for the synthesis of proteins. The preparation also favours calcium deposition in the skeletal bones. The course of therapy with anabolic steroids should not exceed 4 weeks for children. Big doses of the preparation and their prolonged use can cause excessive calcification and growth retardation.

The preparation is available in 1-ml ampoules of a 5 per cent

oil solution for intramuscular administration The preparation should be kept in the dark

*Insulin* is produced by the beta cells of the *pancreatis insulae* (Langerhans' islets) It decreases blood sugar by intensifying glucose metabolism in tissues and conversion of glucose into glycogen Insulin promotes penetration of glucose and the potassium ion into cells The preparation is used for premedication of asthenic patients before operations Insulin is also added to transfused solutions to replenish potassium deficit during operations (12-16 units per 100-150 ml of a 3 per cent potassium chloride) One unit of insulin should be added per each 3-4 g of glucose

Contraindications gastroduodenal ulcer, nephritis, hepatitis

Insulin is available in 5-ml vials (1 ml contains 40 units) The preparation should be kept in a refrigerator Freezing is forbidden.

### Anabolic Non-steroid Preparations

*Potassium orotate* (dioron, oropur) is a potassium salt of orotic acid which is a part of nucleic acids involved in the synthesis of proteins The preparation is considered as an anabolic substance It is used for premedication of patients before operation and during the post-operative period It does not irritate the gastrointestinal mucosa The preparation is used for diseases of the liver, intoxications, heart failure, and for replenishment of potassium loss It potentiates the cardiotonic effect of cardiac glycosides and favours repair processes in the myocardium The preparation is prescribed in a daily dose of 10-20 mg per kg body weight (for 2-3 intakes)

Potassium orotate is produced as 0.5 g tablets There are no special storage regulations

### Plasma Substitutes and Disintoxicating Solutions

*Polyglucin* (dextravan, expandex, macrodex) is a partly hydrolysed dextran in a 0.9 per cent isotonic sodium chloride solution Its molecular weight is close to that of albumin The preparation does not penetrate vascular membranes and when introduced into the blood circulating system remains in it for a long time It is used as a plasma substitute and anti-shock preparation for shock caused by injuries, bleeding or burns It is used to prevent hypovolaemia The preparation quickly raises and stabilizes the arterial pressure in acute blood loss The preparation is slowly (within 1-2 days) withdrawn from the body In cases with severe shock it is injected rapidly, later administration is by intravenous drip

Contraindications cranial injuries with high intracranial pressure, anuria, and heart failure

The preparation is available in 400-ml bottles Should be stored at a temperature of 10-20°C

*Rheopolyglucin* is a 10 per cent colloidal solution of partly hydrolysed dextran containing isotonic sodium chloride solution. The preparation improves the rheological properties of blood, decreases aggregation of the formed elements of blood, and promotes the passage of fluids from tissues to the blood vessels The preparation is used in pathological conditions associated with disturbed blood circulation in the peripheral vessels, for prophylaxis and treatment of shock, and in burns and peritonitis (for detoxicating purposes)

Contraindications: thrombocytopenia, renal dysfunction, heart failure

The preparation is available in 400-ml bottles, should be stored at a temperature of 10-25°C

*Hemodes* is a water-salt solution containing 6 per cent of low-molecular polyvinylpyrrolidone and the ions of sodium, calcium, magnesium, and chlorine It is used in conditions attended by pronounced intoxication of various aetiology (burn disease, infection, peritonitis) The preparation is administered in a dose of 5-10 ml per kg body weight If the preparation is administered rapidly, a moderate fall in the arterial pressure, tachycardia and allergic reactions are possible The preparation is not recommended for use in bronchial asthma, acute nephritis and disordered cerebral circulation

*Placental albumin* is a plasma substitute It is used in hypoalbuminaemia, but its efficacy is more pronounced than that of plasma The preparation increases the oncotic and osmotic pressure to intensify the passage of the interstitial fluid into the blood vessels The preparation is used in burns, shock, nephrotic syndromes, chronic purulent processes, and in brain oedema The preparation is intended for intravenous drip administration

Placental albumin is available as a 5, 10 and 20 per cent solution in 50, 100, 200 and 400-ml bottles

*Lactasol* (Ringer's lactate) is the Ringer solution in which part of sodium chloride is substituted for sodium lactate The preparation is used to decrease the volume of extracellular fluid, especially in pathological loss of liquid from the middle and lower portions of the gastrointestinal tract, for correction of metabolic acidosis, and also as a blood diluent The preparation is administered by intravenous drip (to 1.5-2 litres)

*Gelatinol* is an efficacious plasma substitute useful for dilution of blood during operations on the heart with extracorporeal blood circulation It has no antigenic properties The preparation is effective in acute blood loss, shock and in severe intoxication and infection Protein can be found in the urine in 1-2 days after administration,

because it is excreted by the kidneys in the unaltered form. Contra-indications acute and chronic nephritis

The preparation is available in 250, 300 and 500-ml bottles  
Storage temperature, not above 6°C

### Preparations for Parenteral Nutrition

*Caseine hydrolysate* is the product of acid hydrolysis of caseine. It contains amino acids and simple peptides, and 0.7-0.9 per cent total nitrogen. The preparation is intended for parenteral nutrition of patients before and after operations for replenishment of protein loss.

Caseine hydrolysate is available in 250 and 500-ml bottles.

*Aminosol* is caseine hydrolysate. The protein concentration in solution is 10 per cent. The preparation is widely used for parenteral nutrition of patients of any age.

Aminosol is available in 50, 100 and 400-ml bottles. The preparation should be stored at 20°C.

*Vamine* is a solution of crystalline amino acids (7 per cent), it contains amino acids, fructose (10 per cent), sodium (50 mmole/l), potassium (20 mmole/l), and small quantities of calcium, magnesium and chlorine. The caloric value is 650 kcal/l. The osmotic concentration is 1275 mOsm/l.

*Freamine* is similar to vamine by its properties. It is a 8.5 per cent solution of crystalline amino acids. It also contains 18.3 mmole/l sodium and 30 mmole/l potassium. The caloric value is 340 kcal/l.

*Glucose* is used for detoxication therapy and as a carbohydrate component of parenteral nutrition in 5-40 per cent solutions. The caloric value of a 10 per cent solution is 440 kcal/l. When used for transfusion, it also contains 1 unit of insulin per 4-5 g of glucose.

*Fructose* is a carbohydrate component of parenteral nutrition, though it is used less frequently than glucose. The caloric value of a 10 per cent solution is 380 kcal/l.

*Intralipid* is a 10 or 20 per cent emulsion of soya bean oil in water, egg-yolk phospholipids are used as an emulsifying agent. The osmotically active agent is glycerol. The caloric value of a 10 per cent emulsion is 1100 kcal/l. The osmolarity of a 10 per cent emulsion is 280 mOsm/l, and of a 20 per cent emulsion, 300 mOsm/l.

Intralipid is available in the form of 10 and 20 per cent emulsions in 500-ml bottles.

*Lipofundin* is an oil emulsion similar to intralipid by its organoleptic properties. The essential substance is soya bean oil (10-20 per cent concentration). Soya phospholipids act as an emulsifying agent. The osmotic agent is xylitol. The caloric value of a 10 per cent emulsion is 1100 kcal/l.

Lipofundin is available as a 10 and 20 per cent emulsion in 100-500-ml bottles. The preparation should be kept at a temperature from 2 to 8°C.

### Preparations and Solutions Used for Correction of Acid-base Balance

*Sodium hydrocarbonate* (aqueous solutions with alkaline reaction). The bicarbonate solutions in the concentration of 3, 4, 5 and 7 per cent are used to correct metabolic acidosis in various pathological conditions (shock, intoxication, infection, hypoxia, cardiopulmonary resuscitation, profuse bleeding, and massive blood transfusions). From 10 to 20 ml of a 4 per cent solution of sodium bicarbonate are added to each ampoule of transfused blood (3-4 day-old blood).

Sodium bicarbonate solutions are available in 20 and 50-ml ampoules (3 and 5 per cent solutions).

*Potassium chloride* is mainly used for prophylaxis and treatment of hypokalaemia. It is administered by intravenous drip. The dose depends on the potassium content of the plasma and erythrocytes. The solution is infused together with a 5 or 20 per cent glucose solution and insulin for correction of intracellular potassium deficit. Insulin increases permeability of the cell membrane to promote the passage of potassium into the cell. Rapid administration of potassium chloride can upset heart action to a complete cardiac arrest. Contraindications: severe excretory dysfunction of the kidneys.

Potassium chloride is produced as powder. The preparation should be kept in air-tight containers.

## PART TWO

# Paediatric Anaesthesia

### Chapter 6

## Classification of Anaesthesia

Modern means of anaesthesia have been developed and are quite numerous, and in order to facilitate the selection of the method to be employed, the anaesthetics are classified on the basis of knowledge of the problem of anaesthesia and the clinical requirements.

| General anaesthesia              | Local anaesthesia |
|----------------------------------|-------------------|
| Simple anaesthesia               | Topical           |
| Inhalation anaesthesia           | Surface           |
| Non-inhalation anaesthesia       | Injectable        |
| (a) intravenous                  | Local             |
| (b) intramuscular                | Regional          |
| (c) intravenous                  | Spinal            |
| (d) rectal                       | Perineural        |
| (e) electric                     | Transcutaneous    |
| Combined (a) and (b) anaesthesia | Combined          |
| Inhalation anaesthesia           |                   |
| Non-inhalation anaesthesia       |                   |
| Combined with pre-anaesthetic    |                   |
| Combined with local anaesthesia  |                   |
| Combined with local anaesthesia  |                   |

This classification includes all types of anaesthetic preparations, methods and their combinations.

### ANAESTHESIA COMPONENTS

In order to prevent operation stress, several anaesthetic components are necessary. Depending on the initial condition of the patient and the character of operation, separate or all components may be required.

I Inhibition of psychic perception or rendering patient unconscious. The emotional reactions of a child before an operation are depressed by premedication or basis narcosis. During operation the patient is made unconscious by any inhalation or non-inhalation anaesthetic or by their combination. Depression or inhibition of the child's consciousness for the time of the operation or a painful manipulation is obligatory!

II Central or peripheral analgesia. Central analgesia is attained

by general anaesthetics which act on the central nervous formations involved in pain conduction. All general anaesthetics have good analgesic effect. Most efficacious of them are ether, penthrane, trilen, and halothane. Analgesia can be attained by administration of narcotics such as morphine, promedol, or phentanyl. Peripheral analgesia means depression of perception or conduction of pain impulses by local anaesthetics administered by any suitable route. Peripheral analgesia in general anaesthesia is attained by additional infiltration of local anaesthetics in the receptor fields or by blocking the nervous trunks, which improves substantially the general anaesthetic effect.

**III Neurovegetative blockade** The neurovegetative system is blocked to a certain degree by anaesthetics and analgesics. But a more reliable blockade is ensured by ganglioblocking agents, neuroplegics, central and peripheral cholino- and adrenolytics, which are administered before and during anaesthesia, and also by regional anaesthesia. The preparations lessen excessive vegetative and hormonal reactions of a patient to stress factors arising during surgical interventions, especially in prolonged and vast operations.

**IV Muscle relaxation.** Moderate relaxation of muscles is necessary in practically all operations. This component becomes especially important in operations with artificial lung ventilation. Relaxation of muscles is attained with general anaesthetics whose efficiency however differs. Ketamine is markedly less effective than halothane or barbiturates. Muscles in the operation zone can be relaxed by almost all methods of local anaesthesia (except infiltration). The muscles should be relaxed significantly during operations on the abdominal and thoracic organs. Muscle relaxants, blocking impulses in the neuromuscular synapses, are most suitable for this purpose.

**V Maintaining adequate gas exchange** Gas exchange during operation and anaesthesia depends on the character of the main disease, surgical injury, depth of anaesthesia, accumulation of sputum in the respiratory duct of the child, high carbon dioxide content in the patient-apparatus system, the position of the patient on the operating table, and some other causes. Effective pulmonary ventilation can be ensured by observing the following requirements: 1—correct choice of anaesthesia technique (mask and endotracheal intubation), i.e. spontaneous or controlled respiration of the child during operation, 2—patency of the child's airways, this is attained by a correct position of the patient's head and the mandible during anaesthesia, using airways, periodic aspiration of mucus from the nasal cavity, the mouth and the trachea, 3—correctly selected gas masks, endotracheal tubes, connectors, the entire breathing circuit, and the gas flow-rate. All this is necessary to prevent the increase in the dead space and to preclude hypercapnia or other possible complications.



These requirements hold not only for inhalation anaesthesia but also for all other types of anaesthesia

VI Maintaining adequate blood circulation. Children are sensitive to loss of blood (hypovolaemic conditions) because the compensatory capacity of the heart's pumping function (with respect to the capacity of the vessels, as compared to adults) is low. Maintaining adequate blood circulation therefore requires regulation of the water-salt metabolism and correction of anaemia before operation. Any loss of blood should be replenished completely and in due time during the operation and in the post-operative period. The amount of blood that may be lost by a child during operation is approximately known. Most anaesthesiologists use a gravimetric method for determining the blood loss during operation by weighing the operation materials and bearing in mind that about 55-58 per cent of its weight is blood. The method is very simple but only tentative. It is quite natural that adequate anaesthesia is very important for blood circulation. There are pharmacological preparations that can prevent and treat vascular spasms, arterial hypertension, hypotension and control the tone of the heart muscle.

VII Maintaining metabolic processes. This includes adequate energy supply during operation and anaesthesia, regulation of the water-salt, protein and carbohydrate metabolism, and the acid-base balance, hormone-substitution therapy, regulation of diuresis and the body temperature. All these problems are discussed in detail in the appropriate sections of the book.

### ONE-COMPONENT ANAESTHESIA

One anaesthetic is used to depress consciousness and to ensure analgesia and muscle relaxation. One-component anaesthesia (both inhalation and non-inhalation) is used in minor operations (painful procedures, examinations, or wound dressing). Nitrous oxide (laughing gas), halothane, ketamine, sombrevin, and trilen are commonly used for this purpose. The advantage of this method is its simplicity. The main disadvantage is the high concentration of the anaesthetic causing undesirable side-effects on the bodily organs and systems. Stages of anaesthesia can be well illustrated by using one-component anaesthesia. The classical case is ether anaesthesia. The scheme proposed by Guedel in 1937 (modified slightly by Zhorov in 1959) is popular. And although this type of anaesthesia is not practically used with children, it is useful to describe its stages because other types of anaesthesia are usually compared with the ether model.

The following stages are distinguished: analgesia (I), excitation (II), surgical stage (III), which in turn is subdivided into states III<sub>1</sub>, III<sub>2</sub> and III<sub>3</sub>, and the stage of recovery (IV).

**Stage I.** The analgesic effect increases gradually during the course of 2-3 minutes after inhalation of the anaesthetic. The tactile sensitivity remains unaltered. At later time the consciousness gradually fades and is lost by the end of stage I. The colour of the skin and mucosa does not change, the reflexes remain unchanged, and no substantial changes occur in respiration and blood circulation. The specific ether odour, which intensifies with increasing ether concentration, often provokes cough, suffocation, laryngospasm, and vomiting during the first anaesthesia stage, especially if premedication is untimely or not sufficiently efficacious. Minor surgical operations, e.g. opening of an abscess, can be conducted during the first anaesthesia stage. But it is difficult to stabilize ether anaesthesia during the first stage because as the patient's body becomes saturated with ether, the first stage rapidly passes into the second. If the anaesthetic concentration decreases, the child recovers its senses completely.

The second anaesthesia stage begins when the patient is asleep.

**Stage II** The excitation stage during ether anaesthesia is quite dramatic: the patient develops motor anxiety and groans, the speech is inarticulate. The face reddens, the child tries to remove the gas mask, his pupils are dilated, salivation and lacrimation intensify. The arterial pressure rises, the pulse rate accelerates, the respiration becomes irregular, apnoea alternates with tachypnoea. The reflexes increase. This stage is often characterized by vomiting with trismus of the masticatory muscles.

In the past times, when one-component anaesthesia with ether was widely used everywhere, the anaesthesiologist had to increase ether concentration with the first signs of excitation and to assist lung ventilation to accelerate saturation of the body with ether. The excitation stage was thus shortened and the clinical manifestations were lessened. In at least 20 per cent of cases, the excitation stage is accompanied by vomiting. The mouth of the patient should rapidly be cleaned from the vomit, the gas mask should be changed, and anaesthetizing should be accelerated as described above. This marked excitation is the main disadvantage of ether anaesthesia.

**Stage III** Three substages of the surgical stage are distinguished depending on the depth of anaesthesia. The surgical stage follows the excitation stage and can be stabilized during both shallow and deep anaesthesia. Most operative interventions are conducted during the third stage.

The *first substage* (III<sub>1</sub>) is characterized by even and slightly accelerated respiration, the pulse and the arterial pressure gradually normalize. The eye-balls are fixed eccentrically or move slowly, the pupils are narrow and respond to light, though with a delay. The reflexes (except the corneal and pharyngeal) are depressed. Relaxation is only sufficient to immobilize the patient.

The *second substage* ( $III_2$ ) begins with disappearance of the corneal, pharyngeal and laryngeal reflexes. The eye-balls are fixed centrally, they are moist, the pupils are narrow and respond to light only slightly. Pulse and arterial pressure are normalized, breathing becomes regular and deep. The muscular tone decreases sufficiently to allow operations in the abdominal cavity, except those associated with vast laparotomy.

The *third substage* ( $III_3$ ) is characterized by pronounced relaxation (relaxation of the respiratory muscles included). The respiratory function is maintained mostly by the movement of the diaphragm. This interferes with the surgeon's manipulations in the upper portion of the abdominal cavity. Respiration may become paradoxical: the lower portion of the chest is retracted during inspiration. The eyeballs remain central, the cornea dries up, the pupils contract to a pin-head size and do not respond to light, anisocoria is possible, the eye-slits are slightly open. The pulse accelerates, the arterial pressure increases slightly, the respiration is deep but slow. Further narcotization (it is more difficult to stabilize stage  $III_3$  than stage  $III_2$ ) is fraught with the danger of overdosage and inhibition of the vital bodily functions. The pulse becomes thready and fast, the arterial pressure falls, cyanosis and hypercapnia develop. The eye-balls are soft, the pupils are dilated and do not respond to light, they may have irregular shape. This stage is inadmissible.

**Stage IV.** Supply of ether should be discontinued some 10-15 minutes before the end of the operation. The ether concentration thus begins decreasing gradually. The main portion of ether is eliminated by the lungs. The patient wakes in 15-20 minutes and his condition is reversed, except that the recovery is quiet. The patient is considered to recover from his narcotic sleep only after his consciousness is sufficiently regained. The recovery may be slow if there were some complications during operation: hypoxia, incompletely replenished blood loss, subcooling of the child, overdosage of anaesthetics or analgesics, etc. This period requires special attention on the part of the attending personnel because complications may occur such as chills, vomiting, tongue retraction, loss of coordination of movements (the child may tear off the bandage, fall down from his cot, or injure himself in any other way). The child requires intensive care during 6-10 hours, even after complete recovery from anaesthesia.

The above-described stages are characteristic for anaesthesia induced by any agent. Although the encephalographic picture of a narcotized brain is to a certain extent the same with all anaesthetics, the clinical picture varies significantly. For example, the excitation stage is not pronounced with barbiturates, or it is impossible to attain the surgical stage with nitrous oxide. The stages of ether anaesthesia are therefore only landmarks for the study of any other type of anaesthesia.

A clinical picture of non-inhalation *one-component* (*ketamine*) *anaesthesia* is given below by way of illustration

Stage I begins in 1-2 minutes after the injection. The child gradually calms down and becomes sleepy. He is conscious and correctly (though with difficulty) answers questions, articulation is disordered. The child reacts to pain, i.e. analgesia is not characteristic of this stage. The eye-balls move, and spontaneous vertical or horizontal nystagmus occurs periodically. The pupils are dilated moderately and respond promptly to light. The skin colour does not change. Respiration is spontaneous, undisturbed. Heart rate and arterial pressure do not practically change. The stage lasts for 2-3 minutes and ends with the loss of consciousness. ECG shows reconstruction of the bioelectrical activity: groups of low-frequency waves (mostly of the  $\theta$  range) appear periodically during stimulation of the high-frequency ECG component and  $\alpha$ -rhythm. The ECG can in general be characterized by mixed rhythms.

Stage II begins with the loss of consciousness. The patient sleeps and does not respond to questions. The reaction to pain is very slow. The eye-balls continue moving spontaneously, the pupils are moderately dilated and react quickly to light, the eye reflexes are preserved. The skin dries up and its colour does not change. The heart rate increases by 15-20 per cent and the arterial pressure by 10-15 per cent of the initial. Spontaneous respiration is regular and quiet. The stage lasts for 4-5 minutes and ends when the patient does not respond to pain stimuli (incision of the skin). ECG is characterized by increased amplitude and regular  $\theta$  waves. The alpha-rhythm is preserved while its amplitude slightly diminishes. The waves of the  $\delta$ -rhythm are irregular and with a small amplitude.

Stage III (surgical stage) begins in 8-10 minutes after the intramuscular injection of the preparation and is characterized by the following: the eye-balls are fixed centrally, nystagmus is absent, the pupils are contracted to medium size (do not contract to the point size), they react quickly to light, lacrimation is moderate, and the laryngeal and pharyngeal reflexes are preserved. The skin remains dry and warm, the eye conjunctiva is moist and lustrous with a pronounced injection of the sclera, muscular relaxation is good, spontaneous respiration is normal or it only slows down and becomes deeper, the arterial pressure increased by about 25-30 per cent, and the heart rhythm by 30-35 per cent. By its clinical signs this stage of anaesthesia corresponds to substages  $III_1$  and  $III_2$ , but the transition from substage  $III_1$  to substage  $III_2$  is indistinct. Substage  $III_3$  is not attained with one-component (*ketamine*) anaesthesia. The surgical stage lasts for 25-30 minutes. Low-frequency  $\delta$ - and  $\theta$  waves prevail on the ECG during this stage. The alpha-rhythm appears as single waves with very low amplitude (Fig. 23).

Stage IV (recovery) varies in length from 1 to 4 hours, depending

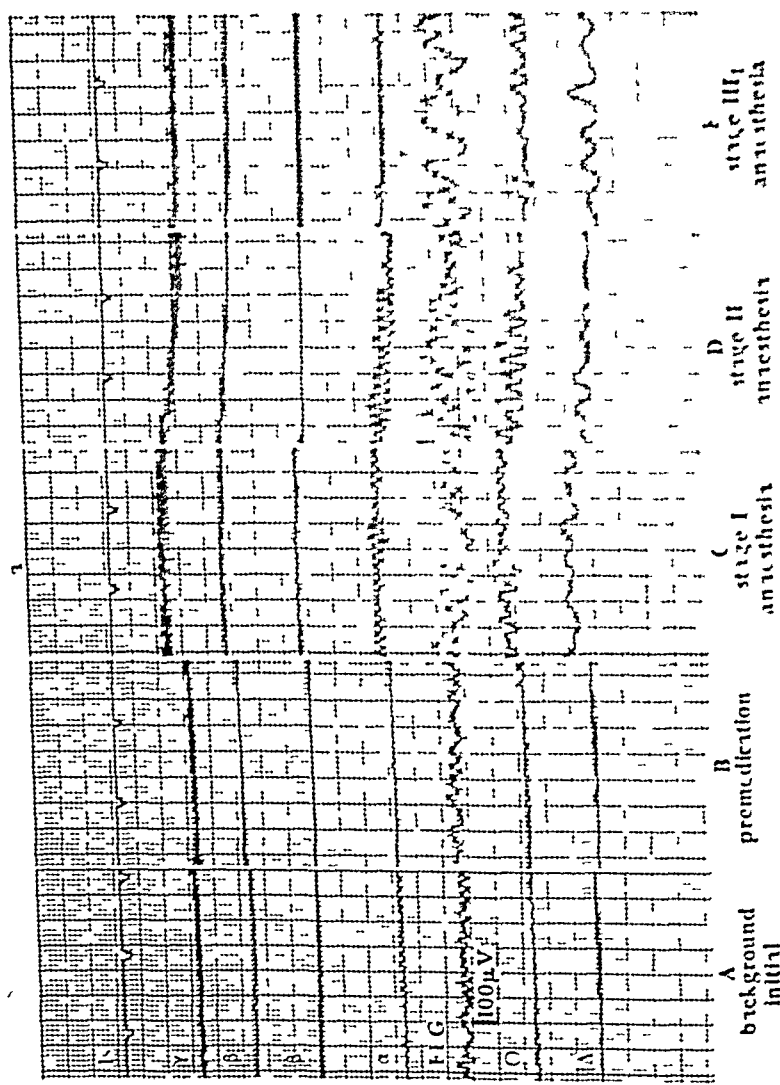
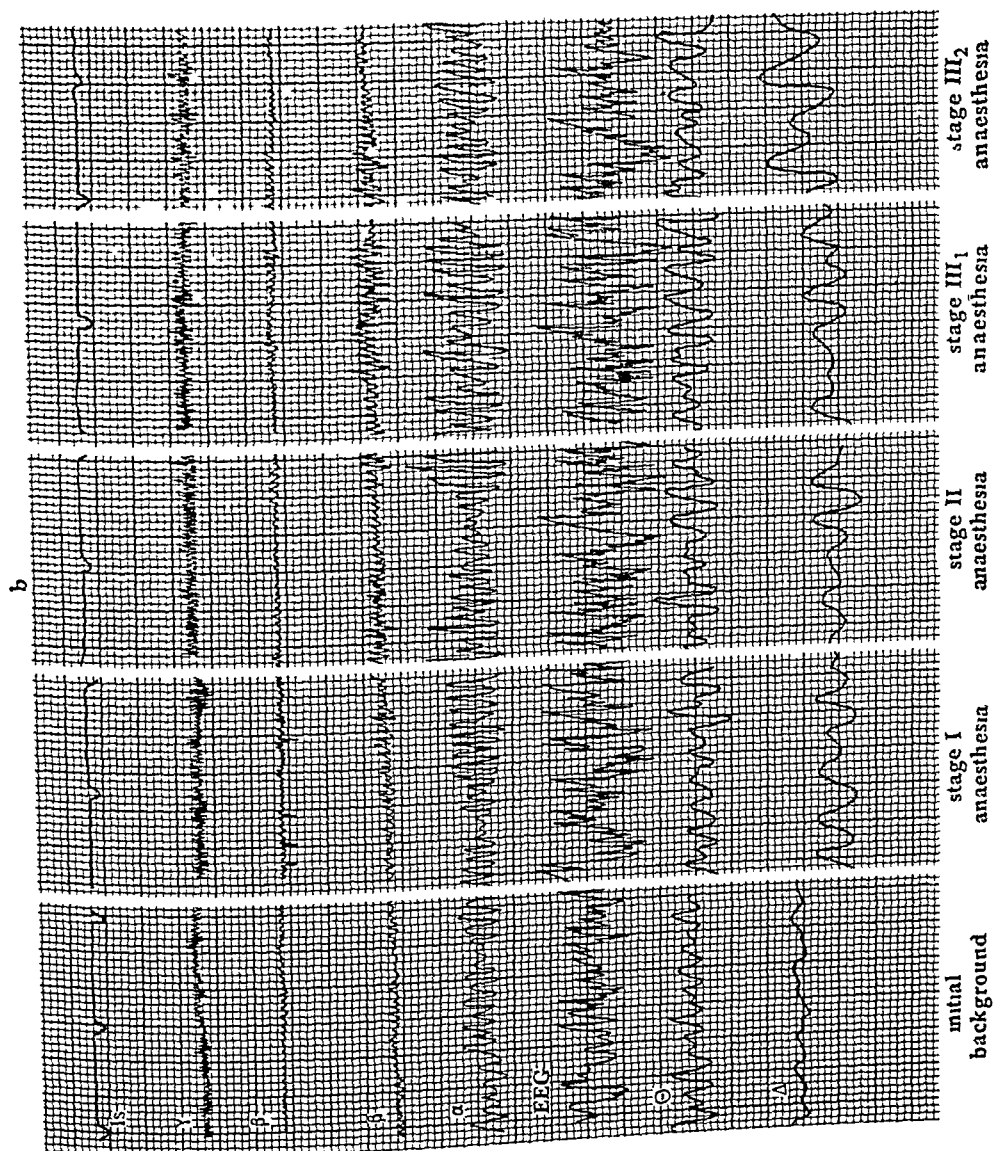


Fig. 23. Electroencephalographic picture of anesthetic states in children  
a—ketamine anesthesia, b—halothane anesthesia



on the time when the last dose was administered, the total duration of anaesthesia, premedication, and also specific properties of a particular patient. During the early post-operative period (3-4 hours) the operated children require analgesia. The clinical picture of stage I and II symptoms is reversed. Excitation, delirium or hallucinations do not occur in children under 10. The ECG is characterized by a prolonged depression of the bioelectrical activity (for 3-4 hours)  $\delta$ - and then  $\theta$ -waves gradually disappear, the amplitude of the fast rhythm increases, i.e. bioelectrical activity becomes non-synchronous. The height of the  $\alpha$ -waves depends directly on the age of the child.

### Inhalation Anaesthesia

Inhalation anaesthesia is most commonly used with children. The anaesthetic is administered as a gaseous mixture into the airways of the child, where the anaesthetic diffuses from the alveoli into the blood and saturates the body tissues. It follows therefore that the desired depth of anaesthesia is attained quicker if the concentration of the anaesthetic in the breathing gas and the minute respiratory volume are high. Solubility of an anaesthetic in the blood and fats is also important. The higher the solubility, the weaker the anaesthetic effect. The main advantages of the inhalation anaesthesia are its easy control and maintenance of the desired anaesthetic concentration in the blood. A relative disadvantage is the necessity of using special apparatus.

Inhalation anaesthesia can be administered through a simple mask or using a mask of a special anaesthetic machine, or by an endotracheal method.

### Non-inhalation Anaesthesia

Anaesthetics of this group are administered by any possible route except through the airways. The most common method is by intravenous injections (barbiturates, sombrevin, sodium oxybate, ketamine, neuroleptanalgesics). The preparations can also be injected intramuscularly. Ketamine is especially frequently administered intramuscularly. Other routes of administration (per os, per rectum, intraosseous) are seldom used. The advantage of the non-inhalation administration of anaesthetics is its simplicity (no special apparatus are required). Non-inhalation method is very convenient for induction of anaesthesia (the period lasting from the moment of administration of an anaesthetic to the time when the surgical stage is attained). The disadvantage of the method is its poor control. Non-inhalation anaesthesia is widely used in minor paediatric surgery and is often combined with other types of anaesthesia.

### COMBINED ANAESTHESIA

Combined anaesthesia means consecutive or simultaneous administration of various anaesthetics and also their combination with other preparations, such as analgesics, tranquilizers or relaxants, which are active components of anaesthesia themselves or potentiate the action of some other anaesthetic components. Various anaesthetics are used in combination with one another in order to attain the maximum effect of a particular preparation, to strengthen the efficiency of some other preparations, and thus attain the desired effects with minimum possible doses. A typical example is the combination of nitrous oxide with liquid inhalation anaesthetics. Nitrous oxide strengthens the weak analgesic effect of halothane. When combined with ether, nitrous oxide ensures a more quiet induction. Inhalation anaesthetics can thus be used together with non-inhalation preparations, non-inhalation anaesthetics can be combined with neurolept-analgesics, etc.

The discovery of muscle relaxants and their use in anaesthesiology have changed the approach to combined anaesthesia. Relaxation of muscles that was formerly possible only with anaesthetics in high (toxic) concentrations can now be attained with muscle relaxants. Adequate anaesthesia can now be attained with relatively small doses of preparations whose general toxicity is low. For example, the patient can be rendered unconscious using sombrevin, his muscles can be relaxed by special muscle relaxants. Analgesia can be attained by administering phentanyl, while anaesthesia can be maintained with small doses of fluothane. Adequate gas exchange can be maintained by artificial lung ventilation.

### Combined Anaesthesia with Muscle Relaxants

Indications for combined anaesthesia with muscle relaxants can be absolute or relative.

*Absolute indications* are as follows

- 1 Operative surgery on the thoracic organs
- 2 Operative surgery on the abdominal organs requiring complete relaxation of the muscles of the anterior abdominal wall
- 3 Operations on the upper airways and neurosurgical operations

*Relative indications*

- 1 Prolonged operations on a patient in physiologically inconvenient positions interfering with normal lung ventilation
- 2 Operations on the face, neck, ear, nose, throat, and some ophthalmological operations

*The conditions for anaesthesia with muscle relaxants*

- 1 Using muscle relaxants during operations on children requires artificial lung ventilation with intubation of the trachea



2 Muscle relaxants should be administered only to a sleeping child, irrespective of his age

It should be remembered that combined anaesthesia with muscle relaxants offers many advantages to the patient, the surgeon and the anaesthesiologist, but it can cause many severe complications. Unreasoned use of combined anaesthesia with muscle relaxants accounts for the increasing number of severe post-anaesthetic complications and disagrees with the principle anaesthesia should not be more injurious than surgery

### CONCURRENT ANAESTHESIA

With this type of anaesthesia the child is rendered unconscious for operation by a general anaesthetic, while a local anaesthetic is used to relax muscles in the operating site, to attain peripheral analgesia, and to block vegetative nerves. Local anaesthesia alone is very seldom used with children, but it is very popular when used in concurrent anaesthesia. The techniques of local anaesthesia are now well developed. For example, epidural anaesthesia and high regional anaesthesia of the extremities have been significantly improved along with improvement of some preparations, e.g. trimecaine, lidocaine, which are now used for epidural anaesthesia instead of procaine and dicaine. Inhalation general anaesthetics should be preferred. Their effect is controlled more easily and they are effective in stabilization of the second and third (III<sub>1</sub>) anaesthesia stages.

The concurrent anaesthesia method can be described schematically as follows: the child is premedicated in the ward, while anaesthesia is induced in the operating room with nitrous oxide and oxygen. Blockade of the operating site is then attained (on a sleeping child), using a suitable local anaesthesia method. After the operation is over, the administration of the anaesthesia is discontinued and the child recovers senses in a few minutes, but the state of hypoaesthesia (in the operating site) persists for a certain length of time. The general condition of a child undergoes smaller changes after concurrent anaesthesia than with other types of anaesthesia.

## Chapter 7

### Preparing a Child for Operation and Anaesthesia. General Principles of Anaesthesia

The anaesthesiologist should be involved actively in preparing a child for an operation and anaesthesia. The preparatory procedures can be divided into therapeutic and pre-anaesthesia, psychological and pharmacological premedication.

## THERAPEUTIC PRETREATMENT

The child, even the youngest child, should be examined by the anaesthetist. The child's condition should be verified, the laboratory tests, such as haemoglobin, should be studied, and the functional state of the respiratory system ascertained. If this is done, additional examination and correction of disorders if any can be planned. The anaesthetist should examine the child 2-3 days before the operation. All drug therapy, pathological findings should be recorded into the child's medical record card.

It is also important to know if the child is important for the anaesthetist in the operation. It is necessary to find out if there is a history of previous labour, if obstetrical forceps or vacuum extractor were used, if there is any history of perforation if the infant had blood transfusion, or if any resuscitation measures were taken. Diseases of the past history of the child should be established (hepatitis, encephalitis, etc., if possible). It is necessary to establish if the infant is susceptible to respiratory viral infections (the time of the last such infection), if he was treated with corticosteroid hormone, and if particular antibiotics were used in the past 10 days. The family history is also important. The anaesthetist should find out if the infant's relatives have any special intolerance of some preparations, e.g. ambroxin, analgin, procaine, etc., because the child may inherit this particular intolerance genetically. It is necessary to find out if the child has any psychic or other disorder that may be important for the anaesthesiologist.

*At a glance at the child.* The agreement between the body weight, height and age, the psychomotor development, obvious defects in the muscle and bones, and behavioural traits give a general impression of the diseased child and the character of possible pathology.

*Nervous system.* With the development of resuscitation technique and further advances in obstetrics, the number of survivals in critical obstetric cases is constantly increasing. Hence, the number of infants with sequelae of labour injuries and diseases of the nervous system, such as encephalopathy, epilepsy, hydrocephalus, paresis of peripheral nerve trunks, etc., increases too. This should be remembered when selecting means for premedication and anaesthesia, because infants are more sensitive to hypoxia and the neuroregulatory compensatory capacity of a child is low. If a child has had Erb's paralysis in his past history, the subclavian vein of the involved side should not be catheterized to prevent possible injury of the brachial plexus.

It is also necessary to assess the condition of the pupils, vision power, the hearing function, possible presence of dysphagia that can be the result of affection of the nervous system. Such children are difficult to communicate with.

*Respiratory system* The condition of the airways of a child is always important for the anaesthesiologist. The following factors should be assessed:

1 The presence of acute respiratory infection. This may cause post-intubation stenosis of the larynx or post-operative pneumonia. Hyperaemia of the fauces alone cannot be a reliable sign of infection because hyperaemia may develop in a child after vomiting and even crying (in neonates). Elevated temperature, rhinitis, and enlargement of the lymph nodes are more reliable signs. The operation should be postponed in the presence of inflammation of the upper airways. If an urgent surgical operation is necessary, prophylactic measures are necessary: antibiotics, inhalation of medicinal solutions, cleaning of the nasal cavity, endotracheal tubes of smaller diameter.

2 Patency of the upper airways. Children often have adenoids, atretic choanae, distorted nasal septum, or the Pierre Robin syndrome, which impair patency of the upper airways to interfere with normal respiration and conduction of anaesthesia. This is especially important for neonates and infants who cannot breathe with the nose. A correctly chosen induction anaesthesia using an airway and a mask for induction of anaesthesia and thorough care of the patient until he completely recovers from anaesthesia can minimize the pathological effect of the disease.

3 Respiratory insufficiency. Atelectases or defects of the lungs that often develop in pneumonia should be timely revealed by the anaesthesiologist. These defects can be suspected during percussion and auscultation of the lungs, and also by the presence of tachypnoea. Chest x-ray makes these defects obvious. If respiratory insufficiency is connected with the main disease for which the operation is to be performed, the anaesthesiologist should establish its degree by determining the blood gases, minute volume (by spirographic and other special methods of examination) and take measures to lessen the insufficiency. This can be attained by sanitation of the bronchi, medical exercises, inhalation of broncholytics by placing the patient in position facilitating drainage, by oxygen therapy, and vibration massage. Antibiotics should be prescribed for acute respiratory diseases of the lungs.

*Cardiovascular system* The agreement between the age of the child and the pulse rate and arterial pressure should be determined. Peripheral circulation should be assessed by the colour of the skin and mucosa, by the difference of skin and rectal temperatures (normally 0.5-0.7°C). In the presence of pathology, ECG should be taken, central venous pressure measured, the gas composition of blood determined, and the cardiothoracic index calculated on the chest x-ray. This information (together with the clinical data) is used to verify the aetiology and degree of the heart failure.

Cardiosurgery requires a greater amount of examinations to be performed. These include echocardiography, probing of the heart chambers with x-ray examination of the heart and measurement of pressure in its chambers, the study of the central haemodynamics by the thermodilution and stain dilution methods. These methods provide comprehensive information on the condition of the cardiovascular system for a correct selection of the anaesthetic method.

The measures aimed at preparing the patient for anaesthesia and operation, and also for lessening the heart failure, include selection of digoxin dose and administration of potassium preparations, ATP, and cocarboxylase. Adrenal cortex hormones and  $\beta$ -antiadrenergic agents are prescribed for indications.

*Abdominal organs and urinary system.* Vomiting and regurgitation during anaesthesia are complications that can cause aspiration of the gastric contents into the airways with subsequent severe complications. The empty stomach before anaesthesia is therefore a prerequisite. The last meal should be given to the child in the evening, on the eve of the operation. If the operation is to be performed in the afternoon, the child may be given to drink a half-cup sweet tea three hours before the operation.

Children are likely to hide sweets and fruits under the pillow, and they readily eat them if no breakfast is given. Besides, food can be given to the child by the ward mates. An infant may cry from hunger and the mother, although being specially instructed, sometimes gives food to her child without being aware of the possible severe complications. A case was reported the mother fed her 3-year-old child an orange an hour before the operation. The child died during induction of anaesthesia. Postmortem section revealed the obstruction of the trachea with an orange section.

If an urgent operation is to be performed, the stomach of the patient should be emptied through a gastric tube. This should be done even if the patient ate for the last time several hours ago, because the evacuatory function of the stomach is strongly upset in some acute surgical diseases of the abdominal organs, much congested fluid may be accumulated in the stomach in the presence of intestinal obstruction or peritonitis. Disregarding this requirement will sooner or later cause complications. It is also necessary for the same reasons to check if the patient has no loose teeth or orthodontic plates. At present researchers work at prophylactic administration of anacid preparations into the stomach before anaesthesia in order to lessen the harmful effects of possible aspiration of the gastric contents. This is especially important in cases where it is impossible to empty the stomach through the tube. An evacuant enema is usually given to the patient before operation.

*Liver and kidneys.* The rate of metabolism and excretion of non-inhalation anaesthetics and relaxants directly depend on the he-

hepatic and renal function. Moreover, inhalation anaesthetics containing the halogens produce a toxic effect on the liver. Liver bleeding during operation may be the result of decreased content of II, V, and VII coagulation factor in the presence of hepatic insufficiency. The functional condition of the liver and the kidneys are therefore very important for the correct selection of anaesthesia. Hepatic failure is usually associated with the pathology of the biliary ducts, portal hypertension, defects of the hepatic artery, and also operations in the presence of septic condition.

Renal insufficiency accompanies pyelonephritis, glomerulonephritis and can develop in pathology of the urinary system. It is easy to suspect these disorders during the study of anamnesis and clinical signs. The degree of disorder can be determined by additional biochemical tests with determination of protein and its fractions, prothrombin, transaminases, urea, creatinine, bilirubin, by the thymol turbidity and Zimnitsky tests.

Hypoalbuminaemia and acidosis are corrected, glucose with insulin, and also vitamins (vitakol included) are prescribed, diuresis is stimulated with lasix (3-4 mg/kg a day).

In the presence of severe hepatic and renal dysfunction, haemoperfusion is sometimes used to decrease the bilirubin content in obstructive jaundice. haemodialysis is conducted when the patient is prepared for transplantation of the kidney.

*Haemostasis* Many diseases of the abdominal organs, the kidneys, lungs and the heart, and post-burn conditions are accompanied with disorders in the water-salt metabolism, protein metabolism, the acid-base equilibrium, and anaemia. Correction of these disorders in the pre-operative period is the main task of the anaesthesiologist. The presence of these disorders depends on the character of the main or concurrent disease, while the degree of disorder is assessed, and the medicines for their correction are prescribed on the basis of the laboratory findings. Deficiency of adrenal cortex hormones can always be suspected in the presence of the adrenogenital syndrome or if the child was treated in the past with hormones (especially with peroral administrations of these preparations). Prednisolone being used as the basis for calculation of the present dose, or the prednisolone dose should be 3-5 mg/kg.

The anaesthesiologist examines the child and gives his conclusion on his condition and the degree of operation risk, prescribes the appropriate treatment before anaesthesia and operation, and makes the appropriate record in the case history. All patients are divided into those who should be operated immediately (emergency cases) and those who were preliminarily examined and assessed as patients who require an operation as a planned procedure. In the former case, the anaesthesiologist must be skilled enough to be

able to decide on urgent operation and to prepare a child in the critical condition for the operation. Or he must be able to decide, together with the surgeon, whether or not a radical operation may be postponed and only a palliative measure be taken at the present time. In the latter case, the anaesthesiologist has the right to postpone the operation till the time when the child is prepared appropriately.

When concluding on the condition of the child before operation, the anaesthesiologist, together with the surgeon, assesses the risk factor, which is a very complicated task. Risk includes many factors on which the outcome of the operation depends: the child's condition, the presence of grave syndromes, the degree of affection of vital functions, the character of the operative intervention, and the age. Of course, skill and experience of the surgeon and the anaesthesiologist, the availability of the necessary apparatuses and the degree of urgency are also included into the notion of risk factor. Various tables have been elaborated by which all factors may be compared and the degree of risk determined. These tables can also be used to foretell the operation outcome. The risk factors should be recorded in the case history.

### PREPARING FOR ANAESTHESIA

The anaesthesiologist's task is to protect the child from negative emotions associated with the operation and to premedicate the child in view of possible complications that may be connected with anaesthesia.

*Psychological treatment* Hospitalization itself is a trial to the child who is separated from his parents and home. The strange environment and the disease promote development of fears. The child may be afraid not only of the people in white, but his moral state depends also on the attitude to him from his ward mates, who can frighten the child by telling their experience, or annoy the child in some other ways. Children are curious and observant. They may examine the trolley on which a patient is transported from the operating room, they may peep into the room where the wounds are dressed, they may be frightened by the sight of patients with drainage tubes, bandaged limbs, etc., and they may live in anticipation of similar experience. The anaesthesiologist must find a correct way to talk to his patient to deserve his confidence and to find out the child's fears unnoticeably. The physician should find out what his patient knows about anaesthesia, he can give the child a gas mask to play with, to try to breathe through the mask, to persuade the child that an injection is not so painful as it might be feared of. Older children should not be deceived. They should be told that they will sleep throughout the operation and will wake in their ward when

the operation is already over. But the child should not be told all truth. Before leaving his patient, the anaesthesiologist must reassure him that none except himself will take him to the operating room. This will quiet the child. After a talk with the child the anaesthesiologist should decide what particular medication should be given before operation and where it should be done (in the ward, in the anaesthesia room, or straight in the operating theatre if the operation should be performed immediately in connection with bleeding or the like).

*Premedication* There are the following general requirements for premedication: 1—psychic comfort of a child, 2—normal salivation, 3—decreased tone of the vagus, 4—potentiation of anaesthetics, substances used for premedication should not inhibit respiration.

The mentioned factors differ in their importance depending on a particular patient. Some manipulations and minor operations do not require decreased tone of the nervus vagus especially so if anaesthesia is conducted with preparations which do not increase salivation. The analgesic component should be increased in some other cases. In some cases the administration of tranquilizers or intramuscular or rectal basis anaesthetics can ensure the required calming effect on the patient's psychic condition.

Many various preparations are now used for premedication. They can be classified in several groups:

1 *M-cholinolytics* These are antisalivary and vagolytic preparations. Atropine and methacine are commonly used for premedication. They have also a slight sedative effect. These preparations prevent bradyarrhythmia that otherwise occurs when depolarizing relaxants are administered, hypersalivation is also prevented by them. Tachycardia and upset thermoregulation in nursing infants are among the adverse side-effects (mostly of atropine). The preparations are administered intramuscularly 45 minutes before anaesthesia (or intravenously immediately before the induction of anaesthesia). Intravenous administration ensures a more stable and reliable effect. Methacine is used in combination with other medicines that are used for premedication in the form of suppositories. For various reasons, other preparations of this group are seldom used.

2 *Hypnotics* Barbiturates (and non-barbiturates) make a large group, but most frequently used are only phenobarbital, aminobarbital sodium, noxiron, and radedorm. Infants sleep well the night before the operation, but older ones (in the pre- and pubertal age) should be given hypnotics. The hypnotics may be given per os (tablets), but if suppositories are available, rectal administration should be preferred. Hypnotics are not as a rule used in the morning before the operation.

3 *Tranquilizers* Having a sedative effect, tranquilizers are widely used for premedication. Vast experience has been gained with

the paediatric use of meprotane, trimetozine and chlordiazepoxide. Parenteral administration of diazepam is most effective because this preparation has a broad therapeutic action with the central relaxation effect and the potentiating effect on analgesics and anaesthetics.

4 Neuroplegic and neuroleptic preparations Dipiazine (pipolphen) and droperidol are widely used for paediatric anaesthesiology. Dipiazine has a marked sedative effect, antihistaminic and broncholytic action, and potentiates the action of analgesics and anaesthetics. It is often used together with atropine and promedol (45-60 minutes before the operation). Droperidol is usually given together with atropine and a strong analgesic phentanyl. This combination ensures very efficacious ataxia and analgesia and slightly inhibits respiration, which is undesirable with spontaneous respiration.

5 Analgesics Promedol is usually given to children. The preparation intensifies the sedative effect of premedication and ensures analgesia during anaesthesia. The inhibiting effect on respiration is lower than that of morphine, nausea and vomiting are also less frequent with promedol. It favourably combines with tranquilizers, neuroleptics and neuroplegics. Phentanyl is a strong analgesic. It is used in combination with droperidol (see above).

Atropine and promedol have remained the most common preparations used for standard premedication. Vast research has shown that this premedication is effective only in children with a strong type of nervous system and ineffective in children with weak nervous system. If diazepam is added (0.2 mg per kg body weight), this premedication proves quite effective in children with both strong and weak nervous systems.

Premedication with atropine and ketamine has a marked analgesic and sedative effect. But tachycardia, hypertension, increased tone, and other side-effects of ketamine have made us look for some other premedication system. We use ketamine in combination with droperidol or diazepam. Premedication with atropine, ketamine and droperidol or diazepam proves efficacious in 94-96 per cent of cases.

The following premedication systems are used: 1—atropine (0.1 mg/kg) + promedol (0.1 mg/kg), 2—atropine (0.1 mg/kg) + ketamine (2.5 mg/kg), 3—atropine (0.1 mg/kg) + ketamine (2.0 mg/kg) + droperidol (0.1 mg/kg), 4—atropine (0.1 mg/kg) + ketamine (2.0 mg/kg) + diazepam (0.2 mg/kg). A particular premedication system should be selected depending on the kind of operation, type of anaesthesia, and the patient's psychic and general condition.

The dependence of doses of common preparations on age is shown in Table 16.

*Routes of premedication* The preparations can be administered per os, intramuscularly, intravenously, or per rectum. Peroral



Table 16 Doses of Preparations for Premedication of Children

| Child age | Atropine | Methacine | Promedol | Diprazine | Barbitamyl | Diazepam | Thalamonal |
|-----------|----------|-----------|----------|-----------|------------|----------|------------|
|           | mg       |           |          |           |            | ml/kg    |            |
| Neonates  | 0.1      | 0.1       | 0.5      | 2-3       | —          | 1-2      | —          |
| to 6 m    | 0.15     | 0.15      | 1        | 5-7       | —          | 3        | 0.06       |
| 6-12 m    | 0.2      | 0.2       | 1.5-2    | 10-12     | —          | 3-5      | 0.06       |
| 1-3 y     | 0.3      | 0.3       | 3        | 15        | 0.01       | 5-7      | 0.05       |
| 4-6 y     | 0.4      | 0.4       | 6        | 20        | 0.03-0.05  | 7        | 0.05       |
| 7-9 y     | 0.5      | 0.5       | 9        | 25        | 0.1        | 8        | 0.04       |
| 10-12 y   | 0.6      | 0.6       | 10       | 30        | 0.15       | 8        | 0.04       |
| over 12 y | 0.8      | 0.7       | 15       | 30-40     | 0.2        | 10       | 0.04       |

administration is the least effective because young children would reject taking tablets, furthermore, vomiting is possible. Operations on the organs of the upper abdomen cannot be performed with peroral premedication. Hypnotic tablets can be given to adolescents before the night sleep on the eve of operation. Intramuscular injections are most common. They guarantee the desired effect of premedication but infants fear injections. Intravenous administration is suitable in urgent cases or when it is necessary to strengthen the premedication effect after anaesthesia has already been started, for example with nitrous oxide or halothane. But the most sparing route of administration of medicines is per rectum. The advantage of premedication per rectum is quick absorption of the active principle of the suppository without irritation of the rectal mucosa. Vast experience has proved the efficacy of rectal administration of medicines to children.

### GENERAL PRINCIPLES OF ANAESTHESIA

There are some general principles that are common for all types of operative surgery and anaesthesia.

**Selecting anaesthetics** The list of anaesthetics includes a great variety of preparations for general and local anaesthesia, and a suitable preparation can thus be selected for each patient with any kind of pathology. But there is no 'ideal' anaesthetic so far. It is difficult to imagine a universal anaesthetic that can be used with equal success, e.g. for correction of hernia in a neonate in conditions of a hospital and a severely wounded soldier in field conditions. A skilled anaesthesiologist follows the principle: a case-specific anaesthetic to each individual patient. Below are the general criteria which should be followed when selecting an anaesthetic and the anaesthesia technique.

- 1 The character of the forthcoming intervention (the amount of surgical intervention, its emergency, and the like)
- 2 Age of the patient and his condition by the moment of induction of anaesthesia.
- 3 The presence of concurrent diseases
- 4 Conditions under which anaesthesia is performed
- 5 Special properties of anaesthetics

Still, a kind of a standard anaesthetic method is often used for minor operations in each particular hospital which however does not contradict the above specified general principles. Nitrous oxide and halothane or ketamine are usually used for minor surgical operations. The rule 'case-specific anaesthetic to each individual patient' does not imply the application of various types of anaesthesia to each particular child. The anaesthesiologist should assess preliminarily the character of surgical intervention and child's condition and to decide beforehand which particular component of anaesthesia is obligatory and which component is unnecessary at all.

The fear of an operation is absent in a neonate or a nursing infant and psychic stress is therefore impossible with them. Their muscular tone is less marked than in older children. The most important anaesthetic component for such patients is therefore adequate analgesia and maintenance of normal respiration and blood circulation. Sparing psychic condition is obligatory for older children. The child should be specially prepared psychologically for any minor operation or manipulation (with appropriate premedication). Anaesthesia should preferably be induced in the ward. Depending on the character of operative intervention and the condition of the patient, special components of anaesthesia should be ensured. Complete relaxation of muscles is obligatory when upper abdomen is operated. Maintenance of adequate lung ventilation and blood circulation is obligatory when the chest of the patient is opened during operation. The general rule that an anaesthesiologist should follow when selecting a particular type of anaesthesia includes using those components of anaesthesia that would minimize the traumatic effect of surgery.

**Transporting a child to the operating room.** The patient should be transported to the operating theatre on a trolley irrespective of the gravity of his condition. The patient should obligatory lie in the trolley because almost all premedication systems include substances having vasoactive properties. Any change in the patient's position may upset his blood circulation and even bring him to a critical condition. Transportation of neonates is a special problem. All care should be taken to prevent exposure of the neonate to cold and to prevent possible regurgitation and aspiration of the vomitus.

Everything should be prepared in the operating room for anaesthesia by the moment the patient is delivered to it.

**Preparing the anaesthesiologist's working post.** This includes checking the availability of all necessary equipment and apparatus and their preparedness for giving anaesthesia, rendering urgent medical aid to the patient, monitoring his condition, and performing the necessary additional manipulations, e.g. bronchoscopy. The condition of the anaesthetic machine should be checked. Checked also should be the store of anaesthetic gases, the condition of the electric aspirator, laryngoscope, bronchoscope, apparatus for artificial lung ventilation, monitoring systems, etc., and also the availability of the necessary set of medicines, sets of tools used for venesection or venepuncture, and systems for transfusion of blood and its substitutes. As a rule, hospitals with anaesthesiological services have reserves of blood and its substitutes that may be urgently required during surgical operations. At other hospitals, special measures should be taken to ensure the necessary blood reserve for a planned operation.

**Composition of the anaesthesiological team.** The anaesthesiological team includes, as a rule, the anaesthesiologist himself and his assistant (anaesthetist). But the team may also be different. For example, an anaesthetist alone can narcotize patients for simple and minor operations (under supervision of some other physician). Or, on the contrary, when the patient is prepared for a complicated operation on the thoracic or abdominal cavity, the team should include another nurse, a transfusiologist, and some other specialists.

**Monitoring the patient's condition.** The patient's condition can be controlled during anaesthesia either by clinical signs or using special apparatus. Pulse rate and arterial pressure are important factors of blood circulation during anaesthesia. An important clinical sign that can characterize the peripheral blood circulation and many other functions is the colour of the skin. Normal diuresis during anaesthesia not only indicates adequate functioning of the kidneys but also adequate renal blood circulation.

Using a simple monitor, which controls the action of the heart during anaesthesia, facilitates the work of the anaesthesiologist considerably. The monitor counts the pulse rate and records (on an oscilloscope screen or on a paper chart) the electrocardiographic findings. More complicated monitors are used to control several physiological functions simultaneously: respiration rate, body temperature, etc. Some modern monitors can control more than 20 physiological functions, such as the cardiac output, peripheral vascular resistance, they can also work together with a computer to facilitate the physician in establishing a correct diagnosis in order to enable him to give appropriate prescriptions and to correct faults.

The *respiratory function* is assessed by clinical and laboratory findings. Depth and rate of spontaneous respiration, the skin colour, and uniformity of lung ventilation are assessed in spontaneous respira-

tion When conducting artificial lung ventilation, its conditions should be selected depending on the particular patient, first by using nomograms (taking into account the age and sex of the patient, his height and weight, and the body surface area), and then (more accurately) during artificial lung ventilation

The acid-base balance is of major importance The *laboratory findings* are quite informative It is important to determine the acid-base balance not only in the capillary blood but also in the arterial and venous blood It characterizes not only the respiratory function but also the transport function of the blood, utilization of oxygen, and elimination of carbon dioxide from tissues

*Electroencephalography* is used for very accurate determination of the depth of anaesthesia This faculty of EEG becomes even more important in anaesthesiology because the assessment of the anaesthesia depth becomes very complicated with modern general multicomponent anaesthesia But EEG is rarely used *per se* in everyday anaesthesiological practice As a rule, EEG is used in clinical studies of new anaesthetics and in other research Of course, this separation by functions is only conventional, for the sake of convenience, and the physician must 'feel' the patient, i.e. to assess the function of all his organs and systems on the whole.

### Principles of Maintenance Therapy During Operation and Anaesthesia

Attaining the necessary components of anaesthesia in each particular case (see Chapter 6) is aimed at maintaining normal life activity of the body. Unconsciousness or depressed psychic perception during operation and anaesthesia is attained by anaesthetics and analgesics The approach to selection of anaesthesia depth has been different throughout the entire history of anaesthesiology It has been proved now that operations should not be carried out with superficial anaesthesia (on the verge of wake), i.e. in the stage of analgesia (analgesic anaesthesia) At least it holds for children In cases where muscle relaxants are used the patient may wake up unnoticeably to the anaesthesiologist, and this may cause severe complications even in the late post-operative period The depth of anaesthesia should therefore be not below the surgical stage III<sub>1</sub>

A complete analgesic effect (absolute relieving of pain perception) is an obligatory condition for any anaesthetic means Pain impulses may cause severe disorders in the blood circulation and other functions, even if the patient is asleep and his muscles are fully relaxed Inadequate analgesia is manifested by tachycardia, increased and then decreased arterial pressure The masticatory and mimic muscles may twitch and the child may perspire Analgesics should therefore be used even in the surgical stage of anaesthesia, a 0.25 per-

cent procaine solution should be injected into the root of the lung, the pleura and the mesenterium before starting manipulations on the reflexogenic zones

Respiration and gas exchange are maintained during operative surgery by inhalation of air or insufflation of oxygen and air together with vapours of anaesthetics, by ensuring free patency of the airways, and by assisted and controlled respiration. It is important to remember that signs of respiratory depression or upset gas exchange, e.g. changes in the colour of the skin and mucosa, changes in the rhythm and respiratory volumes, or acid-base imbalance should be analysed immediately. The causes of these changes should be diagnosed and measures to eliminate them taken. Operation may not be performed in the presence of deviations from the initial respiratory and gas exchange indices.

Normal blood circulation is maintained by adequate analgesia, respiration, infusion therapy, and special preparations acting on the cardiovascular system. During anaesthesia and operation, the energy and the necessary volume of circulating blood should be maintained by infusion therapy. Liquid deficit due to perspiration, eventration of the internal organs, and some other factors should be compensated. A neonate loses to 60 ml of liquid through the skin during an operation, the liquid loss of a one-year-old infant is to 150 ml, and of a 6-year-old child to 240 ml.

The loss of blood during operation can be determined by various methods. The gravimetric one is the simplest: the dressing materials are weighed before and after operation. The amount of blood aspirated is also counted. The loss of blood should be constantly replenished by a slightly larger volume of liquid, the excess amount increasing with the absolute loss of blood. For example, if 10-15 per cent of circulating blood is lost, the volume of the infused fluid should be 20-25 per cent larger than the lost volume, if the blood loss is 20 per cent of the circulating volume, the infused volume should be 30-35 per cent larger than the lost blood. If 25-30 per cent of circulating blood is lost, the loss should be replenished and additional 50 per cent volume added. If the blood loss exceeds 30 per cent, the infused volume should be doubled. Of course, these calculations are only tentative and many other factors should be considered, e.g. condition of the patient before the operation, the amount of surgical intervention, the length of operation, and other factors.

It is not easy to decide what fluid should be used to replenish the blood loss. It has been established recently that the effect of donor blood transfusion is not always positive. There exists a great danger of post-transfusion hepatitis, reactions to albumins, allergic reactions, aggregation of erythrocytes, formation of thrombi in the pulmonary capillaries, and disorders in the blood coagulating and anticoagulating systems. It has been shown that the oxygen carrying

power of donor blood decreases significantly in 3 days after taking. It has also been established that dilution of blood improves micro-circulation, normalizes many haemodynamic processes, and stimulates erythropoiesis. In this connection, the attitude to replenishment of blood in children with an equal volume of donor blood has been revised. A loss of 15 per cent of blood can be replenished by fluids other than blood (plasma extenders). Polyglucin, rheopolyglucin, plasma, and some other solutions are used as blood extenders. If the loss of blood is between 15 and 20 per cent of circulating volume, only 30-40 per cent of the deficit can be replenished with blood, the remaining volume being replenished by blood extender. If the blood loss is more than 25 per cent, the diluent may be 50 per cent of the infused fluid (50 per cent of whole blood + 50 per cent of diluent).

In addition to replenished blood loss the following volumes of fluid should be infused during anaesthesia to compensate for other losses: 6-8 ml per kg body weight per hour to neonates, and 4-6 ml/kg per hour to infants under 1 year of age. The composition of these fluids should be the following (approximately): 5-10 per cent glucose, rheopolyglucin, albumin, etc. the physiological ratio of solutions with and without salts being 1 : 2.

It may be necessary during an operation to administer various medicines improving metabolism and the heart action, e.g. cocarboxylase, sodium hydrocarbonate, calcium chloride (calcium gluconate), hormones,  $\beta$ -blocking agents, panangin.

### Recovery from Anaesthesia

The physician should be prepared to manage various complications that may arise during the patient's recovery from anaesthesia. These may be apnoea, bronchospasm, chills, circulatory disorders, etc. The anaesthesiologist must do everything to prepare the patient for transportation from the operating room. He must ensure stable blood circulation, adequate spontaneous respiration, free patency of the airways, and adequate response to pain stimuli.

Depending on complexity of operation and the amount of surgical injury, the child's age and his condition before operation, the operated child should be transported either to a general surgical ward for post-operative care or into the intensive therapy department. Monitoring the patient recovering after operation and anaesthesia is as important as during anaesthesia. Monitoring is a great help to the nurse in her care of the patient and control of possible complications. Complications of the early post-operative period are usually connected with residual action of the anaesthetics. The most common complications are chills, nausea and vomiting, disorders in blood circulation, psychomotor excitation or,

on the contrary, depression. Depression may be accompanied with severe respiratory distress or complete cessation of respiration. The vital functions of the body should therefore be observed very attentively both during anaesthesia and during recovery from it. An important factor is patency of the airways. Measures should be taken to prevent retraction of the tongue or aspiration of the vomitus.

## Chapter 8

### Simple (One-component) Anaesthesia

Combined anaesthesia is now commonly used but a necessity arises in some cases to carry out a simple one-component anaesthesia. Moreover, the knowledge of the technique and the clinical course of one-component anaesthesia is obligatory for a correct understanding and mastering of the technique for complicated multi-component anaesthesia.

#### INHALATION ANAESTHESIA

Inhalation anaesthesia is now given through a mask and a special apparatus as shown in Fig. 24. A simple mask alone can be used for inhalation anaesthesia only in special cases when anaesthesia is urgent and is conducted under inappropriate conditions.

**Ether-oxygen anaesthesia.** Ether anaesthesia is used very seldom, when other means of anaesthesia are not available. Before giving ether, the patient should be premedicated with atropine and promedol.

The apparatus is prepared for operation. The oxygen flow is adjusted at 6-8 l/min, the safety valve is opened to the full extent to decrease the expiration resistance, and the child is given to breathe pure oxygen for 1-2 minutes. In order to accelerate anaesthesia the absorber can be switched off for 3-5 minutes and the oxygen flow rate decreased to 2 l/min. Slight hypercapnia is thus induced, which promotes a rapid anaesthesia due to intensified pulmonary ventilation and cerebral circulation. The ether supply is now adjusted at the first division of the scale and then, after each 3-5 inhalations, the pointer should be moved through half a division to increase the anaesthetic concentration. As soon as the child gets accustomed to the smell of ether, the oxygen delivery is decreased to 0.5-1 l/min and the safety valve is closed. The ether supply is now increased but if the child coughs, the anaesthetic concentration should be decreased for a short while until the child stops coughing. The ether supply is then increased again (to 7th or 8th division). When the patient shows signs of anxiety, the ether concentration should



Fig 24 Induction of anaesthesia by a face mask

be increased even more in order to terminate this phase as soon as possible. If a child is healthy and strong, the maximum supply of ether continues for 3-7 minutes in older children. As soon as the surgical stage is attained, the pointer should be set at 3 or 5. The concentration of the anaesthetic may be lower (1st or 2nd division on the scale) to maintain prolonged anaesthesia at the required depth. If signs of hypercapnia appear, the oxygen flow-rate should be increased to 6 l/min, while the safety valve should be opened.

The ether supply should be discontinued 10-15 minutes before the operation is over, while the oxygen delivery should be increased to 8-10 l/min. An obligatory condition for normal ether anaesthesia is removal of mucus from the mouth and trachea by aspiration, correct position of the mandible, and assisted respiration (if spontaneous respiration is inhibited).

**Halothane-oxygen anaesthesia** The apparatus should be provided with a special evaporator outside the system where the anaesthetic mixture is circulated. The evaporator should be calibrated for halothane. The oxygen supply should be adjusted at a rate of 6-8 l/min. The child is allowed to breathe pure oxygen for a short time and halothane is then delivered at a concentration of 0.005 l/l (0.5 per cent by volume). The concentration should then be gradually increased (0.0025-0.005 l/l, or 0.25-0.5 per cent v/v per each 4-5 inspirations). When the patient becomes excited, the concentration should be increased by 0.005-0.01 l/l (0.5-1 per cent v/v).



In 3-4 minutes, the halothane concentration should be increased gradually to 0.03 l/l (3 per cent v/v) and then slightly decreased. The patient falls asleep in 2-4 minutes and the surgical stage is attained in 6-8 minutes. The patient is maintained at this stage by halothane at the concentration of 0.015-0.02 l/l (1.5-2 per cent v/v). As soon as the surgical stage is attained, the oxygen supply is adjusted at a suitable rate, depending on the particular apparatus used (closed or semi-closed circuit). To-and-fro or semi-open systems are recommended to children under 6, while common rebreathing circuits can be used with older children.

It should be remembered that the onset of the surgical stage (contracted and centrally fixed pupils, relaxed muscles, slightly accelerated respiration) does not indicate that the operation can be started. If a surgical incision is made immediately after the onset of the surgical stage, the child may move involuntarily, the pupils may widen and laryngospasm occur. The test for pain reaction should therefore be first conducted. In the presence of response to pain anaesthesia should be deepened. Depending on the length of anaesthesia, the patient may wake up in 4-6 minutes after suspension of halothane supply. The oxygen delivery should be increased during this period to facilitate elimination of the anaesthetic and carbon dioxide from the lungs.

**Halothane-air anaesthesia** Special apparatuses calibrated for halothane should be used. As with the other anaesthetic machines, the halothane supply should first be adjusted at the minimum level of 0.005 l/l (0.5 per cent v/v). During the following 2-3 minutes the halothane concentration should be increased to 0.03 l/l (3 per cent v/v). The patient falls asleep in 2-3 minutes, the surgical stage follows in another 3-4 minutes. The surgical stage is maintained by halothane supplied at a rate of 0.015-0.02 l/l (1.5-2 per cent v/v). The patient may develop hypoxia due to respiratory distress. Halothane-air anaesthesia is therefore rarely used with children.

### Nitrous Oxide Anaesthesia

Adequate premedication is especially important with this type of anaesthesia. Atropine, analgesics (promedol or omnopon), hypnotics, and (for special indications) antihistaminics should obligatorily be used for premedication. Children should be transported to the operating room half-sleeping. Anaesthesia can be given with any suitable apparatus provided with flow-meters for oxygen and nitrous oxide. The system is filled with oxygen. In order to denitrogenate the patient, he is first given pure oxygen to breathe for 2-3 minutes through a mask from the apparatus (semi-closed circuit), the oxygen flow rate being 6-8 l/min. The absorber may be disconnected. The oxygen supply is then decreased to 2 l/min and nitrous oxide

is supplied at a rate of 6-8 l/min. It is necessary to ensure a tight fitting of the gas mask to the child's face. At the oxygen to nitrous oxide ratio of 1 : 4 the child falls asleep in 60-90 seconds, and in some cases after 10-12 inspirations. The gas supply should be maintained at this level for another minute or two, the oxygen to nitrous oxide ratio should then be decreased to 1 : 3.

If the initial oxygen to nitrous oxide ratio is 1 : 3 and the gases are supplied at a rate of 2 and 6 l/min, respectively, the analgesic stage is attained within the first 2-3 minutes. The heart rate decreases insignificantly in children over 6, consciousness becomes dimmed, and the child develops oral and motor excitation. The child becomes unconscious completely in 4-6 minutes from the start of inhalation. If the gas flow rate is decreased, the absorber should be connected to the system. Inhalation of a gas mixture containing less than 0.2 l/l (20 per cent v/v) oxygen even for a short period of time is prohibited.

The surgical stage (III<sub>1</sub>) of anaesthesia is attained in 6-8 minutes from the start of inhalation. The onset of this stage is characterized by a slightly accelerated respiration, tachycardia, live corneal and pupillary reflexes, the presence of normal tone of the skeletal muscles, and slight hyperaemia of the face. In most cases it is impossible to deepen this stage without increasing the anaesthetic concentration. Nitrous oxide is absorbed in a child at a faster rate than in adults. If anaesthesia is only superficial, the child develops motor excitation and arrhythmical respiration. Extremely deep anaesthesia is manifested by cyanosis. Stage III<sub>1</sub> can be maintained in children with the oxygen to nitrous oxide ratio of 1 : 3. This ratio may be lower (1 : 2) with asthenic patients, but this concentration of anaesthetic only maintains analgesia in most cases. If cyanosis and other signs of respiratory insufficiency are obvious, the oxygen delivery should be increased and the anaesthetic effect of nitrous oxide should be intensified by a more effective agent. When the operation is close to completion (3-5 minutes before termination of operation), the oxygen to nitrous oxide ratio should be decreased to 1 : 2 and 1 : 1. Just before terminating the operation, the nitrous oxide supply should be discontinued and the child should be given pure oxygen to breathe. Breathing air after discontinuation of delivery of the oxygen-nitrous oxide mixture is not allowed. It can cause diffusion hypoxia owing to supply of great amount of nitrous oxide from the blood into the lungs.

The child usually wakes up in 3-5 minutes after discontinuation of nitrous oxide supply. Anaesthesia with nitrous oxide does not practically affect the respiratory function, circulation of blood, or consciousness, coughing is absent.

### Trichloroethylene Anaesthesia

Trichloroethylene (trilen) given in narcotic doses causes arrhythmia and disorders in the respiratory function and is therefore used only for short operative procedures it produces only shallow analgesia and the surgical stage is not attained. No special premedication is necessary with trilen anaesthesia but if a child is overanxious, he may be given analgesics. Trilen may not be given from apparatus employing chemical absorbers of carbon dioxide, since trilen will decompose giving a poisonous substance dichloroethylene (ethylene dichloride). Open or semi-open systems can therefore only be used with trilen.

Vaporizers used for trilen anaesthesia are provided with a batching device delivering trilen at a rate of 0.006-0.009-0.015 l/l (0.6-0.9-1.5 per cent v/v). The patient is given trilen by mask, the anaesthetic being inhaled together with atmospheric air. The patient expires into the atmosphere through a special valve.

The patient does not feel any unpleasant sensations when he breathes trilen-air mixture. Analgesia is usually attained after 12-16 inhalations (sometimes even earlier). Consciousness is lost only in 2-4 minutes and the gas mask may then be removed. The patient recovers in 30-40 seconds after discontinuation of delivery of the anaesthetic.

### Cyclopropane Anaesthesia

Premedication should obligatory include atropine. Vomiting should be prevented by intramuscular injections of aminazine. Adrenaline should not be used because cyclopropane sensitizes the myocardium to catecholamines.

The child is given pure oxygen to breathe for 1-2 minutes and then cyclopropane is supplied (closed system) at a rate of 0.25-0.3 l/min, oxygen is delivered at a rate of 1 l/min. The concentration of cyclopropane should be increased to 50 per cent (0.5-0.6 l/min of oxygen and 0.5-0.6 l/min of cyclopropane) within few minutes. After breathing this mixture of equal volumes of cyclopropane and oxygen for 1-2 minutes, the child usually falls asleep and the surgical stage of anaesthesia is attained. If the surgical stage should be attained as soon as possible, the 1:1 mixture can be supplied from the very start of anaesthesia. The patient falls asleep in 1-1½ minute and the surgical stage is attained in another 1-1½ minute. In order to maintain this stage, a mixture containing the cyclopropane is delivered at a rate of 0.1-0.15 l/min while oxygen is delivered at a rate of 1 l/min. Cyclopropane supply should be discontinued 5-7 minutes before the end of the operation, while the oxygen supply should be increased to 6-8 l/min. The patient recovers from anaesthe-

sia in 20-40 minutes. Disorders in blood circulation are possible, and cyclopropane is therefore usually used only for induction anaesthesia.

### Ethrane Anaesthesia

No special premedication is necessary with ethrane (enflurane) anaesthesia. Antihistaminics and other preparations are prescribed for special indications (depending on the condition of the child). The child first breathes pure oxygen for 1-2 minutes and then ethrane is delivered, whose concentration is gradually increased by 0.005 l/l (0.5 per cent v/v) per each 4-5 inspirations. The induction of anaesthesia is quiet because the anaesthetic has a pleasant odour, nor does it irritate the mucosa of the upper airways. The initial stage (I) of anaesthesia ends with loss of consciousness and in children it lasts for 20-90 seconds. The child remains sensitive to pain during this stage. The transitional stage (II) of ethrane anaesthesia lasts from 2 to 10 minutes. The pupils are rapidly contracted, then the ocular and laryngeal reflexes are decreased, the muscle tone lowers, and the sensitivity to pain is finally lost. The excitation stage is practically absent.

The surgical stage is attained in 6-12 minutes from the beginning if the concentration of the anaesthetic is 0.035-0.04 l/l (3.5-4 per cent v/v). Spontaneous respiration is inhibited significantly and the lungs should be ventilated artificially. The heart rate slightly increases, the arterial pressure decreases by 10-15 mm Hg. In order to maintain the surgical stage, the ethrane concentration should be decreased to 0.025-0.03 l/l (2.5-3 per cent v/v). Since the anaesthetic is rapidly eliminated from the body, its concentration should be decreased not earlier than the surgical wound is sutured. The child fully recovers from anaesthesia in 5-10 minutes after discontinuation of the anaesthetic delivery. Since the analgesic effect of ethrane is short, post-operative analgesia should be started on the operating table.

### NON-INHALATION ANAESTHESIA

An obligatory component of medication is atropine.

**Intravenous barbiturate anaesthesia.** Intravenous barbiturate anaesthesia is conducted with a 1 per cent thiopental sodium or hexenal solutions. The barbiturate solutions should be prepared immediately before administration. To that end, 1 g of the dry substance is dissolved in 20 ml of isotonic sodium chloride solution or distilled water in a vial. The solution is then transferred by a syringe into a measuring cylinder and 80 ml of isotonic sodium chloride solution or distilled water are added to make a 1 per cent barbiturate solution. It should be mixed thoroughly by pulling it into the syringe and

ejecting it from the syringe several times. The first portion of the barbiturate solution (2-3 ml) should be injected slowly, within 15-20 seconds. This dose does not usually cause any noticeable confusion of consciousness or inhibit respiration. Pain at the site of injection may be due to injection of the barbiturate solution either under the skin or into the artery. If there is no adverse reaction to the injection, the injection should be continued in 30-40 seconds at a rate of 1 ml during 5-10 seconds. From 7 to 10 ml of the barbiturate solution should be injected within 1-2 minutes. The child falls asleep, the respiratory function is inhibited and the pupillary reaction to light decreases. The corneal reflex remains alive and the eyeballs are mobile. As soon as consciousness is lost, the child should be given oxygen to inhale through the mask. Subsequent administration of another 6-8 ml of the barbiturate solution within 40-60 seconds ensures the surgical stage of anaesthesia (III<sub>1</sub>-III<sub>2</sub>). This state is characterized by a greater inhibition of spontaneous respiration, flaccid corneal reflexes, and immobility of the eyeballs. The operation can be started.

Anaesthesia is maintained by fractional slow injection of the barbiturate solutions (2-3 ml) so that the narcotization remains at the same level. The depth of anaesthesia should be assessed not by the administered dose but by the clinical signs. Motor reactions, tachypnoea or coughing in response to the surgical intervention indicate insufficient depth of anaesthesia. Marked respiratory distress, cyanosis, dilatation of the pupils, relaxation of the muscles and tongue retraction indicate excessively deep anaesthesia (stage III<sub>3</sub>). The administration of barbiturates should be discontinued and assisted or artificially controlled respiration should be induced. If special anaesthetic apparatus is not available, the patient's mandible should be pulled back and artificial lung ventilation should be conducted using an air bag or carrying out the mouth-to-mouth artificial respiration. When anaesthesia is stabilized at the surgical stage, it is unnecessary to administer additional doses of the barbiturates until the first signs of insufficient depth of anaesthesia develop. As a rule, the patient begins recovering in 2-3 minutes after suspension of injections (in 5-6 minutes, if the injections were multiple). The recovery lasts for 3-10 minutes. It is characterized by a gradual recovery of reflexes, motor activity and consciousness. Intravenous barbiturate anaesthesia should be used for induction of anaesthesia only, or if anaesthesia lasts not more than 20 minutes.

**Intramuscular barbiturate anaesthesia.** Promedol should preferably be used for premedication. A 10 per cent hexenal or thiopental sodium solution (1 g of the dry substance dissolved in 10 ml of an isotonic sodium chloride solution or distilled water with subsequent thorough stirring of the solution) is injected into the thigh or the buttocks. The dose is 8-10 ml of a 10 per cent solution

Hexenal or thiopental sodium becomes effective in 3-5 minutes. The child becomes sleepy in 6-8 minutes and a narcotic sleep is induced in 15-20 minutes and lasts for 15-30 minutes. Respiration can be inhibited during this period. Less frequently it occurs due to overdosage and more frequently due to tongue retraction. Assisted lung ventilation is then necessary. Deep anaesthesia is induced by intramuscular injection of barbiturates in weak and asthenic children. If a child is healthy and strong, stage III<sub>1</sub> anaesthesia is only attained. After recovery from anaesthesia, children fall asleep again for 30 to 120 minutes.

This type of anaesthesia is seldom used because of its poor control.

**Intramuscular ketamine anaesthesia** Atropine and diazepam should obligatory be used for premedication. Narcotic analgesics, antihistaminics and some other preparations can also be used for special indications.

A 5 per cent ketamine solution is used for intramuscular injections. The anaesthetic dose depends on the age and the body weight of the patient. For neonates the dose is 12-14 mg/kg, for nurslings it is 10-12 mg/kg, infants ageing from 1 to 2, 9-11 mg/kg, from 3 to 6, 8-10 mg/kg, and for children ageing from 7 to 14, 7-9 mg/kg. The lighter the child, the bigger the anaesthetic dose with reference to his body weight. The rate of ketamine absorption after its intramuscular injection depends on the dose and the local condition of tissues. Absorption is rapid from the muscles of a child with adequate blood circulation, and slow if the circulation is inadequate or if the solution is injected into fat. Like in any other type of general anaesthesia, the individual response to ketamine depends on the dose and the age of the child. An allergic reaction may develop at the site of injection (hyperaemia or macular eruption).

The main dose of the anaesthetic is administered 8-10 minutes before operation. A single intramuscular injection ensures a strong analgesic effect and the patient falls asleep. In other words, a single dose ensures induction and basal anaesthesia. The maximum effect is attained in 8-10 minutes, this period is marked by pronounced tachycardia and hypertension. One dose is effective for 25-30 minutes. If an operation requires a longer time, surgical anaesthesia should be maintained by repeated intramuscular injections of ketamine. If signs of shallow anaesthesia develop in 25-30 minutes after the first injection, either the initial dose is injected again or the dose may be halved, depending on the anticipated time of operation. The anaesthetic stage should be maintained by repeated intramuscular injections at 20-25-minute intervals ( $1/2$  or  $1/4$  of initial dose).

Ketamine injections should be repeated for the following indications: *a*—accelerated respiratory rate and decreasing tidal volume,

*b*—obvious motor reactions of the limbs, *c*—twitching of the mimetic muscles, *d*—displacement of the eyeballs and nystagmus. The last dose of the anaesthetic should be administered 30-40 minutes before the end of the operation.

Recovery from anaesthesia lasts from 30 to 60 minutes and depends on the time when the last dose was administered, the time of general anaesthesia, preparations of premedication, and special properties of the patient.

Ketamine has a pronounced analgesic effect and no additional analgesics are therefore required during 2-3 hours post-operative.

**Intravenous ketamine anaesthesia** A 1 per cent ketamine solution (5 per cent ketamine diluted in 5 per cent glucose to obtain the ketamine concentration of 1 per cent) is used for intravenous administration. The main dose is 2-3 mg/kg, irrespective of the patient's age. The maximum effect is attained in 40-60 seconds and lasts for 10-15 minutes. This period is characterized by elevated arterial pressure and marked tachycardia. Anaesthesia is maintained by repeated intravenous injections of the preparation at 10-15-minute intervals ( $\frac{1}{2}$  or  $\frac{1}{4}$  of initial dose). If the anaesthetic is injected at a fast rate (within 60 seconds), the respiratory function of children is strongly depressed to a complete cessation of respiration, artificial lung ventilation is then needed.

The patient recovers from anaesthesia more rapidly than with intramuscular injections, but still the time of recovery varies between 15 and 30 minutes.

## Chapter 9

### Multi-component Anaesthesia

#### NITROUS OXIDE AND HALOTHANE ANAESTHESIA

Atropine should be used for premedication. The patient is first given oxygen to breathe for 1-2 minutes at a rate of 6-8 l/min. The nitrous oxide to oxygen ratio in the gas mixture is 3 : 1. Halothane delivery is started simultaneously. The concentration is increased gradually from 0.005 to 0.025-0.03 l/l (from 0.5 to 2.5-3 per cent v/v). If the oxygen supply should for any reason be increased, the nitrous oxide proportion should be decreased to 50 per cent (and even lower) increasing the concentration of halothane. The surgical stage is attained without excitation in 2-3 minutes. The initial nitrous oxide to oxygen ratio can then be restored or adjusted to 2 : 1 (3 : 1), while the halothane concentration should be decreased to 0.005-0.01 l/l (0.5-1 per cent v/v). Sometimes physically strong children require the halothane concentration of 0.1-0.015 l/l (1-1.5 per cent v/v) to maintain the surgical stage of anaesthesia. The supply

of halothane should be discontinued 8-15 minutes and of nitrous oxide, 3-5 minutes before termination of the operation (depending on duration of the operation)

### NITROUS OXIDE, BARBITURATE, AND HALOTHANE ANAESTHESIA

Premedication should obligatory include atropine, the other components of anaesthesia should be included for special indications. Adrenaline should not be used. Barbiturates should be used for induction anaesthesia. After the patient falls asleep oxygen inhalation should be carried out for 30-40 seconds (6-8 l/min). The nitrous oxide to oxygen ratio is then adjusted at 2 : 1 (3 : 1), halothane delivery is started at a rate of 0.005 l/l (0.5 per cent v/v). The halothane concentration is gradually increased within 2-3 minutes till the moment when the surgical stage is attained. If the patient shows signs of excitation, a small dose of barbiturates can be administered additionally. The desired stage of anaesthesia is maintained by delivering nitrous oxide and oxygen at a ratio of 2 : 1 and halothane at a rate of 0.005-0.015 l/l (0.5-1.5 per cent v/v). Halothane delivery should be discontinued 5-10 minutes before the operation is completed (depending on the duration of the operation). The oxygen delivery should meanwhile be increased to 40-50 per cent. Nitrous oxide supply should be stopped 3-5 minutes before the end of the operation, while the oxygen supply should be increased to 8-10 l/min.

### KETAMINE AND NITROUS OXIDE ANAESTHESIA

Atropine is an obligatory component of premedication, diazepam is given for indications. The main dose of a 5 per cent ketamine solution is administered intramuscularly. The dose depends on the child's age and his weight: neonates are given 10-12 mg per kg body weight, nurslings, 9-11 mg/kg, infants from 1 to 2 years of age, 8-10 mg/kg, between 3 and 6, 7-9 mg/kg, and between 7 and 14, 6-8 mg/kg. After the anaesthetic is administered, the child is given pure oxygen to breathe for 1-2 minutes and then nitrous oxide is added (the nitrous oxide to oxygen ratio, 3 : 1 or 2 : 1). The surgical stage of anaesthesia is attained in 6-8 minutes, the main dose remains effective for 30-40 minutes. The anaesthesia is maintained by inhalation of nitrous oxide and oxygen at a ratio of 2 : 1 and repeated injections of ketamine ( $\frac{1}{2}$  or  $\frac{1}{4}$  of initial dose). Nitrous oxide eliminates muscular hypertone and ensures a more uniform narcotization. During the maximum effect of the main ketamine dose, moderate tachycardia and hypertension develop. The last dose of the anaesthetic should be injected 40-50 minutes before the end of the operation. The supply of nitrous oxide should be discontinued 5 minutes before



the end of the operation, and the child should be given pure oxygen to breathe for a few minutes

If a 1 per cent ketamine solution is administered intravenously, induction begins with inhalation of nitrous oxide and oxygen at a ratio of 3 : 1. The main ketamine dose (2 mg/kg, irrespective of the child's age) is injected slowly into the vein in 3-5 minutes. The surgical stage is attained in 30-40 seconds and lasts for 15-17 minutes. Anaesthesia should be maintained by inhalation of nitrous oxide and oxygen (2 : 1) and repeated intravenous injections of  $\frac{1}{2}$  or  $\frac{1}{4}$  of initial ketamine dose at 15-20-minute intervals. The last dose of the anaesthetic should be administered 20-30 minutes before the end of the operation.

### KETAMINE NITROUS OXIDE AND HALOTHANE ANAESTHESIA

Premedication is the same as with the ketamine and nitrous oxide anaesthesia. The child is first given pure oxygen to breathe for 1-2 minutes (6 l/min). Then nitrous oxide is added (2 : 1). Ketamine is injected intramuscularly or intravenously in 3-5 minutes. The main dose of the anaesthetic for intramuscular administration is 8-10 mg/kg for neonates, 7-9 mg/kg for nurslings, 6-8 mg/kg for children from 1 to 2 years of age, 5-7 mg/kg for children between 3 and 6, and 4-6 mg/kg for children ageing between 7 and 14.

The main ketamine dose for intravenous injections is 1.5-2 mg/kg. The surgical stage of anaesthesia is attained in 4-6 minutes and it lasts for 50-60 minutes with intramuscular injections (30-40 seconds and 15-20 minutes, respectively, with intravenous injections). The anaesthesia is maintained by injections of  $\frac{1}{2}$  or  $\frac{1}{4}$  of initial ketamine dose (at 1-hour intervals with intramuscular or at 15-20-minute intervals with intravenous injections), and by inhalation of 0.006-0.008 l/l (0.6-0.8 per cent v/v) halothane and a mixture of nitrous oxide and oxygen (2 : 1). This type of anaesthesia stimulates the heart's action though not markedly. At the height of action of the main dose, the rise of the arterial pressure is more uniform and slow. The last dose of the anaesthetic is injected intramuscularly 1 hour before the end of the operation (30 minutes before the end of operation with intravenous administration). The supply of halothane is discontinued 10-15 minutes and of nitrous oxide, 5 minutes before the end of the operation.

### ANAESTHESIA WITH CENTRAL ANALGESICS

#### Nitrous Oxide, Halothane, and Promedol Anaesthesia

Premedication is standard. Anaesthesia is induced by inhalation of nitrous oxide and oxygen (2 : 1) and 0.01-0.015 l/l (1-1.5 per cent v/v) halothane. When the patient is asleep,  $\frac{2}{3}$  total dose of the analgesic is slowly injected intravenously. The total promedol dose

is 0.8-1 mg/kg. If promedol is injected rapidly, spontaneous respiration is strongly depressed to complete arrest for short periods of time, lung ventilation should be assisted.

The surgical stage of anaesthesia is attained in 30-40 seconds after administration of promedol. The respiration becomes slow and deep. The eyeballs are fixed centrally, the pupils are contracted to the size of a point. Changes in the blood circulation are not pronounced. Analgesia is complete and muscular relaxation is adequate. The desired anaesthesia is maintained by inhalation of nitrous oxide and oxygen (2 : 1) and 0.006-0.008 l/l (0.6-0.8 per cent v/v) halothane. If clinical signs of inadequate analgesia develop, the remaining 1/3 total promedol dose should be injected intravenously in 50-60 minutes after the initial injection. The halothane supply should be discontinued 20-30 minutes before the end of the operation. The supply of nitrous oxide should be stopped when placing skin ligatures. The recovery from the anaesthetic sleep is long. Children remain inhibited for 30-40 minutes.

### Nitrous Oxide and Pentazocine Anaesthesia

Atropine and preferably diazepam should be used for premedication. The child is first given pure oxygen to breathe for 1-2 minutes (6 l/min) and then nitrous oxide is added to adjust the nitrous oxide to oxygen ratio at 2 : 1. In 5-7 minutes diazepam is injected intravenously (0.3-0.5 mg/kg). The main dose of pentazocine is injected slowly into the vein within 40-60 seconds (immediately after injection of diazepam). The main analgesic dose depends on the weight and age of the child: 3-2.5 mg/kg for infants from 1 to 2 years of age, 1.5-2 mg/kg for children from 3 to 6, and 1.2-1.5 mg/kg for children from 7 to 14.

If pentazocine is injected rapidly, spontaneous respiration may be inhibited to complete cessation and artificial or assisted lung ventilation may be required. An isotonic sodium chloride solution or a 10 per cent glucose solution should therefore be used to dilute or a 10 per cent glucose solution should therefore be used to dilute pentazocine (1 : 1) before administration. The surgical stage (III<sub>1</sub>-III<sub>2</sub>) is attained in 2-3 minutes after injection of the analgesic, the stage is characterized by moderate hypertension and tachycardia. Spontaneous respiration is not inhibited. The analgesic effect of a single dose of pentazocine lasts for 40-50 minutes. The anaesthesia should be maintained by inhalation of nitrous oxide and oxygen (2 : 1) and by repeated intravenous administration of the analgesic at 35-40-minute intervals ( $\frac{1}{2}$  or  $\frac{1}{4}$  of initial dose). The last pentazocine dose should be administered 40-50 minutes before terminating the operation. The recovery is rapid. Nitrous oxide delivery should be stopped immediately after the operation is over. The child recovers in 2-3 minutes after breathing pure oxygen.

## ETHIRANE AND NITROUS OXIDE ANAESTHESIA

Atropine should obligatory be used for premedication. The child should first be given oxygen to breathe for 1-2 minutes and then nitrous oxide is added to oxygen in the ratio of 2 : 1. Ethrane is added to the mixture in 1-2 minutes and its concentration is gradually increased. Anaesthesia is induced with ethrane concentration of 0.025-0.03 l/l (2.5-3 per cent v/v). The surgical stage of ethrane anaesthesia is attained in 6-10 minutes from the start of induction. The surgical stage is maintained by inhalation of the gas mixture containing 0.02-0.025 l/l (2-2.5 per cent v/v) ethrane and nitrous oxide and oxygen (2 : 1). The supply of ethrane and nitrous oxide should be discontinued when skin ligatures are placed. The child recovers in 5-7 minutes after discontinuation of the anaesthetic supply.

## ATARALGESIA

Ataralgesia is attained by the combined use of sedatives, tranquilizers (ataractics) and analgesics. Consciousness is inhibited completely (with combined use of the above-mentioned agents) with small doses of hypnotics (usually nitrous oxide). Diazepam is the most popular ataractic, while pentazocine, phentanyl, dipidolor, and some other preparations are used as analgesics.

Premedication includes intramuscular administration of atropine and diazepam (the doses depend on age). Nitrous oxide is added to oxygen after the patient breathes pure oxygen for 1-2 minutes. Venepuncture and catheterization of the vein should be conducted with inhalation of nitrous oxide and oxygen (3 : 1). Diazepam is then injected intravenously in a dose of 0.3-0.5 mg/kg. The preparation is first diluted to 10-20 ml with isotonic sodium chloride solution and then injected within 1-2 minutes. The effect is immediate: the eyes move involuntarily or the gaze becomes fixed. The patient turns sleepy and inhibited (ataraxia). The respiration slows down, the arterial pressure decreases by 10 mm Hg. The pulse does not practically change. The child soon falls asleep. The excitation period is practically absent. The clinical course of induction anaesthesia with ataralgesia is similar to the clinic of neuroleptanalgesia, except that the respiration and blood circulation are less affected. The analgesic is administered intravenously immediately after loss of consciousness. The surgical stage is attained in 1-2 minutes after the administration of the analgesic. The surgical stage is maintained by inhalation of nitrous oxide and oxygen (2 : 1) and by repeated administrations of the analgesic.

The necessity of repeated injections of diazepam arises only when the operation continues longer than for two hours. A dose of 0.2-

0.3 mg/kg would usually be sufficient in such cases. The recovery period is rather short. The supply of nitrous oxide is discontinued during placing skin ligatures. The child recovers in 5-10 minutes after suspension of nitrous oxide inhalation.

## Chapter 10

# Multi-component Endotracheal Anaesthesia

## INDICATIONS FOR ENDOTRACHEAL ANAESTHESIA

There are absolute and relative indications for endotracheal anaesthesia in children.

### *Absolute indications*

- 1—operative surgery on the thoracic organs,
- 2—operative surgery on the upper abdominal organs,
- 3—neurosurgical operations and plastic surgery in the mouth,
- 4—operative surgery on patients in physiologically inconvenient postures (prone position, on the side, and the like) during which lung ventilation becomes inadequate,
- 5—urgent surgical operations on the abdominal organs in neonates

### *Relative indications*

- 1—prolonged surgical operations (lasting over 2 hours),
- 2—short operations on the face and neck during which there is a danger of obstruction of the airways,
- 3—urgent surgical operations (prevention of aspiration of the stomach contents)

## TRACHEAL INTUBATION

Direct laryngoscopy is commonly used in anaesthesiological practice for intubation of the trachea. The intubation technique for children differs from that for adults in some details. Two methods are mainly used for intubation of children: the orotracheal method (through the mouth) and the nasotracheal one (through the nose). Laryngoscopy and intubation are usually conducted during apnoea caused by muscle relaxants.

**Orotracheal intubation.** The head of the child is placed over a ring made of cotton wool and gauze or over a small pillow (2-4 cm thick) and deflexed to the maximum extent in the atlanto-occipital junction. In some cases the head is only slightly pulled back. It is then slightly raised by the right hand (put under the shoulders and the neck of the child), while the left hand is used to pull the head back. The lips of the child are now separated and the mandible slightly pulled upwards. The laryngoscope should be held in the left hand and the blade introduced into the mouth, the tongue being

pulled upwards and slightly to the left (Plate 3) A straight blade should be used with children, but a curved one can also be used provided the operator has experience with using this instrument. As soon as the blade rests against the wall of the oropharynx the round entrance to the oesophagus becomes obvious. The laryngoscope is now slightly pulled back and the epiglottis (a dark-pink structure) becomes visible, in children it is more horizontal than in adults. When the epiglottis is pressed upwards, the entrance to the vocal slit opens. If the laryngoscope is passed into the mouth slowly, first the uvula and then the epiglottis can be seen. If a straight blade is used, the free edge of the epiglottis is pressed upwards. The curved blade of the laryngoscope is first brought to the epiglottis base and the epiglottis is then moved upwards. The entrance to the larynx appears as a narrow vertical slit. The movement of the left hand during laryngoscopy should be directed upwards and from the operator, as if raising the child's head. As soon as the vocal slit is seen in the vision field, the endotracheal tube should be passed into the slit to the depth of 1.5-2 cm. In order to keep a constant vision of the vocal slit, the assistant should press slightly the laryngeal wall with his fingers. If intubation is conducted without anaesthesia, or with anaesthesia but without muscle relaxants, the vocal cords close and open during inhalation and exhalation. If the child is medicated with muscle relaxants, the vocal slit is open. The endotracheal tube is passed into the trachea not by the blade groove but at an angle to the blade. After the tube has passed into the trachea, it is held in place (at the lips of the child) by the fingers and the free end of the tube is connected to the apparatus for anaesthesia. The anaesthesiologist compresses the bag of the apparatus by the other hand to conduct artificial ventilation of the lungs.

The correct position of the tube in the trachea is manifested by even excursions of both sides of the chest, checked by auscultation of both lungs (uniform air passage) and by the appearance of the child (pink lips, ear lobes and finger tips). Once the tube has been inserted correctly, it is fixed to the patient's face with two adhesive tapes. During the time when correctness of the tube position and its fast securing is checked, the anaesthesiologist holds the tube in place with his fingers because even an insignificant displacement of the tube can change its position: the tube may enter the right bronchus or enter the mouth cavity. If the tube is moved too far to enter the right bronchus, the left side of the chest is not involved in the respiratory act (or lags in its movements). When the air bag is pressed, the resistance is quite obvious, the child becomes restless, and develops moderate cyanosis and pallor. If the tube end is in the mouth or the oesophagus, the air bag is easy to compress and a specific whistling sound can be heard as the gas mixture passes into the mouth, the stomach may inflate appreciably. The optimum length

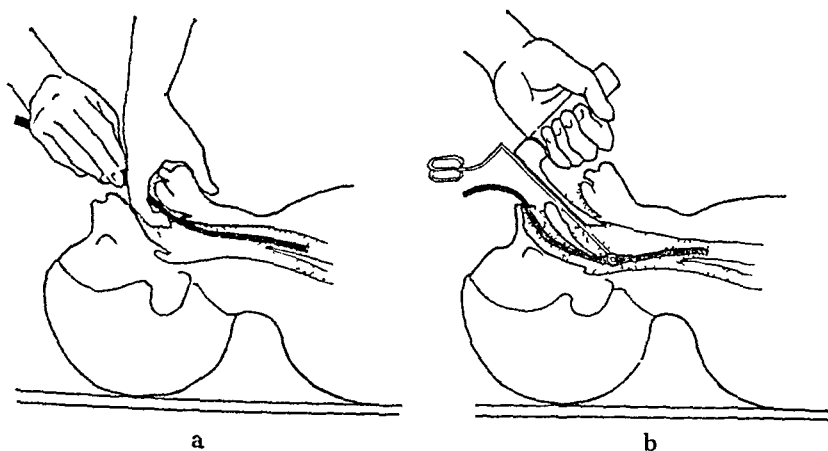


Fig 25 Intubation with guidance of the tube by finger (a) and using laryngoscope (nasal intubation) (b)

of the tube emerging from the mouth is 3-4 cm. Intubation of infants can be done without a laryngoscope, using a finger only (Fig 25a).

*Nasotracheal intubation* It is used in the presence of tumour or abscess in the mouth and also in all cases where the endotracheal tube in the mouth interferes with the surgeon's manipulations. The tube should be oiled with a lubricant containing hormones and passed with anaesthesia through a wider nostril into the pharyngeal lumen. Using a laryngoscope to control manipulations, the tube end is taken by the Magill forceps in the pharynx and directed through the vocal slit into the trachea (Fig 25b). For convenience it is recommended to hold tube with the forceps at a distance of 1-1.5 cm from its end. An ordinary curved tube without inflatable cuff is used for nasotracheal intubation. It is fixed in place so that its free end protrudes from the nose to a distance of 2-3 cm.

#### USING MUSCLE RELAXANTS IN MULTI-COMPONENT ANAESTHESIA

The main principles of using muscle relaxants are as follows:

- 1 Muscle relaxants can be used only in sleeping children.
- 2 Muscle relaxants can be used only with simultaneous artificial lung ventilation, because alveolar hypoxia develops even from their small doses that do not inhibit appreciably spontaneous respiration. The external respiration should be compensated until spontaneous breathing is completely restored.
- 3 Muscle relaxants ensure only relaxation of the muscles and partly hyporeflexia. All other components of anaesthesia, such as inhibi-

tion of consciousness, maintenance of adequate gas exchange, blood circulation, and metabolism, should be attained by other means of modern anaesthesia. Their use is even obligatory since muscle relaxants mask inadequacies of anaesthesia.

The advantages of multi-component anaesthesia with muscle relaxants are as follows:

a—optimum conditions are provided for artificial lung ventilation, which is especially important for operations attended by disorders in external respiration (operations on thoracic organs).

b—the toxic effect of narcotics on the body is diminished due to their smaller total doses. The muscles are relaxed by the muscle relaxants,

c—the airways remain unobstructed in any position of the patient, the possibility of asphyxia due to tongue retraction or aspiration of vomitus or blood is thus excluded, the conditions for constant removal of the tracheal contents are provided,

d—the conditions for gas exchange are improved due to diminished dead space,

e—pressure supply of anaesthetic gas ensures maximum saturation of the body with oxygen and expansion of the lungs.

*Anaesthesia with depolarizing muscle relaxants.* Nowadays depolarizing muscle relaxants are used in children for the following indications: 1—for intubation of the trachea (bronchi), 2—for broncho- and oesophagoscopy examinations with anaesthesia, 3—for anaesthesia lasting for less than 30 minutes with artificial lung ventilation, 4—for lengthy operations in children with renal failure.

Atropine is an obligatory premedication component, while other medicines are used for special indications. Anaesthesia can be induced with any anaesthetic, the choice depending on the initial condition of the child. As soon as the patient is asleep, depolarizing muscle relaxants are injected intravenously (lyshtenon, myo-relaxin, 2-3 mg/kg). Depolarizing muscle relaxants cause random contractions of the skeletal muscles (fibrillation). During this period in connection with inhibited spontaneous respiration inhalation of anaesthetics is halved and assisted lung ventilation conducted. If spontaneous respiration is arrested, the anaesthetic supply is discontinued and the lungs are ventilated artificially with oxygen which is given by mask of the anaesthesia apparatus (moderate hyperventilation). The trachea should be intubated only after complete cessation of fibrillation, because intubation can otherwise be ineffective or injurious. After intubation of the trachea has been performed (with relaxation of muscles), the child is given the anaesthetic mixture to breathe through artificial lung ventilation apparatus. Muscles remain relaxed due to repeated administration of fractional doses of the relaxant at 5-7-minute intervals. The majority of children develop moderate bradycardia, which lasts for 15-60 seconds, after

each administration of the relaxant. The arterial pressure sometimes falls. Prolonged apnoea does not always indicate the efficacy of muscle relaxation because the apnoea in children is often the result of hyperventilation of the lungs, while the muscle tone restores to interfere with surgeon's manipulations. If means of objective control of artificial myoplegia are not available, muscle relaxants should be administered when signs of muscular tone reappear. In prolonged operative interventions, the relaxants should be administered at greater intervals. The last dose of the relaxant should be administered 20-30 minutes before the end of the operation.

Depolarizing muscle relaxants can be used practically with all anaesthetics. The overall dose of muscle relaxants should be diminished with halothane anaesthesia and their administration should be done at greater intervals, because halothane inhibits spontaneous respiration and prolongs apnoea.

*Anaesthesia with non-depolarizing muscle relaxants* Non-depolarizing relaxants are used during operations lasting more than 60-90 minutes. The preparations of this group are used to maintain myoplegia during operation. One dose is effective for 30-40 minutes. These preparations ensure stable and lengthy relaxation of the muscles. The initial dose of *d*-tubocurarine chloride for children is 0.2-0.3 mg/kg. Since the preparation has cumulative properties, each next dose should be  $\frac{1}{3}$  smaller. Clinical indications for repeated administration of non-depolarizing muscle relaxants are the following:

- 1 Increased resistance to inhalation (assessed by resistance to compression of the air bag)
- 2 Development of muscular strain in the abdominal wall
- 3 Development of spontaneous respiration after cessation of artificial lung ventilation
- 4 Development of the coughing reflex to the introduction of an endotracheal tube
- 5 Convulsive movements of the diaphragm characteristic of hiccup.
- 6 Movements of the limbs
- 7 Reappearance of potential on an electromyogram (to 50 per cent of the initial magnitude)

The combined use of non-depolarizing relaxants with various anaesthetics is characterized by some special features. When used together with ether, effectiveness of some relaxants increases. It holds to a lesser degree for halothane and cyclopropane. The relaxants of this group can decrease arterial pressure by 10-20 mm Hg and they should therefore be used especially carefully in children with hypotension, during halothane anaesthesia, and in neurolept-analgesia.

*Anaesthesia with depolarizing and non-depolarizing relaxants* An obligatory component of premedication is atropine. Anaesthesia is induced by any inhalation or non-inhalation anaesthetics. After



the patient is asleep, depolarizing muscle relaxants should be administered (lysθενон, myo-relaxin, 2-3 mg/kg). To prevent muscular fibrillation in children small doses of non-depolarizing relaxants (1-3 mg of tubarine) should be given. If spontaneous respiration is arrested, hyperventilation with oxygen is recommended for a short time. If muscles are relaxed (after cessation of fibrillation), the trachea of the child should be intubated and artificial lung ventilation with the anaesthetic mixture is conducted. The depth of anaesthesia should correspond to stage III<sub>1</sub>, i.e. the patient must sleep and analgesia must be adequate. In 5-8 minutes after reappearance of signs of muscular tone, non-depolarizing muscle relaxant (tubarine, 0.2-0.3 mg/kg) should be administered intravenously. A dose of tubarine remains effective for 30-40 minutes. Muscles should be maintained relaxed by fractional administration of tubarine, whose each next dose should be diminished by  $\frac{1}{3}$ . At the end of operation, the dose of the relaxant should be so calculated as to provide conditions for restoration of spontaneous respiration. The last tubarine dose should therefore be administered 40-50 minutes before the end of the operation. Anaesthesia should preferably be deepened rather than the non-depolarizing relaxants administered. Depolarizing relaxants can be used after the administration of non-depolarizing muscle relaxants. Depolarizing relaxants may be administered only after the action of non-depolarizing relaxants has terminated, which is manifested by the reappearance of deep spontaneous respiration and muscular strain. The anaesthesiologist should however remember that the effect of the relaxants may be perverted (inadequate, or on the contrary, excessive myoplegia).

*Use of muscle relaxants without tracheal intubation* Muscle relaxants can be used without tracheal intubation, as a component of the general multi-component anaesthesia. It broadens the anaesthesiological possibilities and facilitates the control of anaesthesia depth and muscle relaxation. Fractional intravenous administration of small doses of relaxants ensures more effective relaxation of muscles, immobilization of the child under shallow anaesthesia, and quickly arrests coughing and laryngospasm. Succinylcholine preparations, which have the lowest inertia of their action, are most suitable relaxing components of general anaesthesia without intubation. Anaesthesia is effected by intravenous administration of barbiturates or by inhalation of nitrous oxide with halothane and oxygen. Succinylcholine is used in a dose of 0.5-1.2 mg/kg. The minimum doses should first be tried, and if they prove ineffective, they should be increased. When spontaneous respiration is inhibited, lung ventilation should be assisted. In cases of apnoea, artificial lung ventilation should be given through the mask.

General anaesthesia with muscle relaxants without tracheal intubation can be indicated for short surgical operations and mani-

pulations requiring adequate muscle relaxation (repositioning of bone fractures, correction of joint dislocation, etc) Using muscle relaxants without intubation requires very high skill on the part of the anaesthesiologist

*Control of child's condition after administration of muscle relaxants*  
This is based on the determination of depth of anaesthesia and the degree of muscle relaxation The visual assessment of the clinical course of multi-component anaesthesia with muscle relaxants is very difficult because the main sign, characterizing the depth of anaesthesia in children (character of spontaneous respiration), is absent At the same time, multi-component anaesthesia with muscle relaxants is not characterized by the classical picture of the one-component anaesthesia Two stages of anaesthesia, shallow and deep, are therefore differentiated in multi-component anaesthesia today

Shallow anaesthesia is characterized by the reaction of the pupils to light and lacrimation After discontinuation of action of muscle relaxants, the clinical picture becomes similar to that of the one-component anaesthesia, in other words, the specific stages can be distinguished in the pupillary reflexes, reactions to pain, etc Excessive sweating and tachycardia, increased arterial pressure, excessive lacrimation, motor reactions to pain indicate insufficient depth of anaesthesia

Deep anaesthesia is characterized by the absence of pupillary reaction to light and other stimuli, upset blood circulation, and depressed vegetative nervous system The determination of concentration of anaesthetics in the inhaled gas mixture and electroencephalographic findings are very important for the assessment of the anaesthesia depth

In addition to assessment of anaesthesia depth, efficacy of muscle relaxants (myoplegia) should also be determined But the determination of relaxation of the skeletal muscles is difficult because the relaxants are always used together with anaesthetics, which have a certain myoplegic effect themselves, their effect is sufficient to mask the true effect of muscle relaxants Myoplegia can be assessed by several methods

1 Palpatory and visual determination of muscular relaxation is one of the most effective methods It is mostly used by the surgeon who assesses the tone of the anterior abdominal wall muscles Restoration of the muscular tone after an operation is determined visually and by palpation

2 Respiratory method This method of determining the effect of muscle relaxants is not reliable and cannot be recommended for wide use, because muscle relaxants have different effect on various groups of muscles and the respiratory function can be affected by some other factors of anaesthesia (hyperventilation, anaesthetics or analgesics, broad-spectrum antibiotics, etc)

3 Determining concentration of muscle relaxants in the blood. There are biological, chemical, spectriographic, and polarographic methods for the determination of muscle relaxants in the blood but they are rather labour-consuming and are not widely used.

4 Electrophysiological determination of effect of muscle relaxants (myography). Muscle relaxants relax the muscles through their effect on the neuromuscular synapse. Therefore myographic determination of the functional condition and conduction of the neuromuscular synapse gives reliable information on efficacy of muscle relaxants.

Cessation of anaesthesia and recovery of the patient are the most important periods of multi-component anaesthesia with muscle relaxants. It is important that the patient wakes up as soon as possible after termination of the operation, while the analgesic effect should last to complete recovery and during the early post-operative period. It is desirable that the child wakes up and regains his respiratory function and protective reflexes on the operating table.

The period of recovery from anaesthesia with muscle relaxants is characterized by some special features. The absence of the clinical picture of respiratory insufficiency and also the values of  $PO_2$ ,  $pH$  and  $PCO_2$  are the signs of adequacy of spontaneous respiration. Despite the decreased doses and timely administration of the last dose of the muscle relaxant, spontaneous respiration in children is often restored slowly. This is one of the most frequent side-effects of muscle relaxants. There exist many reasons of slow restoration of spontaneous respiration after operations, muscle relaxants are not always the main cause of this delay. The most frequent causes are as follows:

1 Hyperventilation during artificial respiration causing hypocapnia. The action of the respiratory centre is restored slowly if the  $PCO_2$  of blood is very low.

2 Upset acid-base balance. This factor is especially important when depolarizing muscle relaxants are used. The acid-base imbalance during anaesthesia is usually characterized by metabolic acidosis. Depolarizing muscle relaxants are slower hydrolysed in an acid medium and their action therefore lasts for longer time. The excretory function of the kidneys is also decreased in metabolic acidosis. This is another factor slowing down the restoration of the respiratory function after operation.

3 Effect of anaesthetics or other preparations on the neuromuscular conduction. This mostly holds for inhalation and non-inhalation anaesthetics, which are used together with muscle relaxants. The neuromuscular block deepens if broad-spectrum antibiotics, analgesics, procaine, and similar medicines are used.

4 Overdosage or excessive accumulation of muscle relaxants in the body. This is a less frequent cause, which should however be al-

ways remembered. Overdosage of muscle relaxants is manifested by a complete absence of the muscular tone and spontaneous respiration, and by complete or partial block of the neuromuscular synapse as recorded on an EMG.

*Decurarization* Proserine (neostigmine, prostigmine) is used as an antidote to non-depolarizing muscle relaxants. Neostigmine lessens the effect of muscle relaxants by inhibiting cholinesterase (as a result of which acetylcholine accumulates and displaces relaxants from the receptors) and by the direct strengthening of the neuromuscular conduction. Antidotes to muscle relaxants are indicated for respiratory distress and low muscular tone in the end of the operation. If a child is able to raise his head or clamp his fist, the muscular tone is considered sufficient. Proserine can be used as an antidote in prolonged respiratory distress and the changed character of block caused by diacetylcholine. This condition is manifested clinically by a delayed restoration (20-40 minutes) of spontaneous respiration.

*Methodology of using proserine* Atropine is first administered intravenously in a dose of 0.5-1 mg. The atropine premedication is obligatory to remove the vagotonic effect of proserine. The pulse rate should accurately be observed for 2-2½ minutes, and then proserine is slowly (within 20-30 s) injected into the vein in a dose of 1-1.5 mg (2-3 ml of a 0.05 per cent solution). If the first dose produces no effect in 2-3 minutes, the injection can be repeated. The effect of proserine is manifested by development of elements of spontaneous respiration.

The antidotes do not exclude a thorough control of the child's condition, mainly his respiratory function. In 30-40 minutes, when the proserine effect discontinues (while the concentration of relaxants in the blood is still high), the muscles can relax again (recurarization). Galanthamine is also used in children.

## SCHEMES FOR MULTI-COMPONENT ENDOTRACHEAL ANAESTHESIA IN CHILDREN

### Anaesthesia with Barbiturates, Nitrous Oxide, and Muscle Relaxants

Atropine premedication is obligatory. Other premedication components should be used for special indications. Anaesthesia should be induced by intravenous administration of a barbiturate (1 per cent hexenal or thiopental sodium solution). The trachea should be intubated after administration of depolarizing relaxants (lysthenon, myo-relaxin) in a dose of 2-3 mg/kg (in the presence of complete relaxation of the muscles). The child is then given artificial lung ventilation with a mixture of nitrous oxide and oxygen. Nitrous oxide is supplied at a rate of 2-3 l/min and oxygen 1 l/min (2.1 or

3 1) A semi-closed system should be used. The absorbers should obligatory be incorporated into the system. If a child is asthenic, the concentration of nitrous oxide may be decreased to 50 per cent. This concentration is insufficient for strong children and arterial pressure in them rises during operation along with acceleration of the pulse rate, when the action of muscle relaxants is over, the extremities begin moving. Anaesthesia should then be deepened by increasing the concentration of nitrous oxide or by intravenous administration of 1.5-2 ml of a 1 per cent promedol solution.

Fractional administration of depolarizing relaxants should be begun immediately after the appearance of shallow spontaneous respiration through the tracheal tube or if a reaction to the endotracheal tube appears. Depolarizing relaxants are usually administered during the first half of the operation at 6-8-minute intervals (the same dose as the initial). During the second half of the operation the effect of the relaxants increases and the dose can therefore be diminished, while the intervals between subsequent injections increased. The last dose of the muscle relaxant should be injected 15-20 minutes before the end of the operation. Spontaneous respiration is completely restored in 8-12 minutes after the last injection, provided the preparation has a common effect on the patient.

### Anaesthesia with Halothane, Nitrous Oxide. and Muscle Relaxants

Atropine and promedol should be used for premedication, while other preparations should be used for special indications. The child is first given pure oxygen to breathe for 1-2 minutes. Then nitrous oxide is added to the breathing mixture (2 l), and finally halothane, whose concentration is gradually increased from 0.005 l/l (0.5 per cent v/v) to 0.02-0.025 l/l (2.0-2.5 per cent v/v). As soon as the patient is asleep, depolarizing muscle relaxants (lysthenon or myorelaxin) are injected in a dose of 2-3 mg/kg, with subsequent tracheal intubation. Then the lungs are ventilated artificially with the gas mixture in which the nitrous oxide concentration is decreased to 50 per cent. The concentration of halothane is decreased to 0.01-0.015 l/l (1.0-1.5 per cent v/v).

Depending on the extent of the operative intervention and the time of operation, the desired level of muscular relaxation should be maintained by fractional injections of both depolarizing and non-depolarizing relaxants. If the operation is long and the operative injury is significant, the non-depolarizing relaxant tubarine should be injected, provided the variations in the arterial pressure during halothane inhalation are insignificant. If the depolarization relaxant was used for intubation of the trachea, no repeated injections of the preparation are required (after discontinuation of its action).

If the patient reacts to the tracheal tube, anaesthesia should be deepened for a short time. After spontaneous respiration is restored, tubarine should be administered intravenously in a dose of 0.2-0.3 mg/kg. This situation usually occurs in 5-8 minutes after the first administration of the depolarizing relaxant. The action of tubarine lasts for 30-40 minutes. During operation relaxation is maintained by fractional administration of tubarine, each time decreasing the dose by  $\frac{1}{2}$ . The analgesic effect of halothane is insignificant and it is desirable that small doses of analgesics (20-30 mg of promedol) should be administered during operation's most dramatic stages. The last tubarine dose should be administered 30-45 minutes before the end of the operation so that spontaneous respiration might be restored by the end of operation. The halothane supply should be discontinued 3-5 minutes before terminating the operation, while nitrous oxide should be excluded from the breathing mixture during suturing the wound. Proserine should be administered to children in whom respiration is not efficient during the post-operative period.

### Anaesthesia with Ethrane, Nitrous Oxide, and Muscle Relaxants

An obligatory component of premedication is atropine, while other components depend on the child's condition. The patient is given pure oxygen to breathe for 1-2 minutes before anaesthesia. Induction into anaesthesia begins with inhalation of nitrous oxide and oxygen (2:1), and ethrane is added to the breathing mixture from a special vaporizer located outside the circulation system. The ethrane concentration should be gradually increased to 0.02-0.025 l/l (2-2.5 per cent v/v). As soon as the patient is asleep, depolarizing relaxants are injected intravenously in ordinary doses and the trachea is intubated. The lungs are ventilated artificially with a mixture of gases in which the concentration of ethrane decreases to 0.01-0.015 l/l (1-1.5 per cent v/v), while the nitrous oxide to oxygen ratio is set at 2:1 or 1:1. Narcotic analgesics (20-30 mg of promedol) should be administered during the operation's most dramatic periods. Relaxation of muscles during the operation is maintained by intravenous fractional administration of either depolarizing relaxants (in short operations and minor manipulations) or non-depolarizing relaxants (in prolonged and injurious operations). The action of relaxants of both types is about the same. The last dose of depolarizing relaxants should be administered 15-20, and of non-depolarizing relaxants 30-45, minutes before the end of operation. The concentration of ethrane in the breathing mixture should be decreased only when wound suturing is started. The supply of nitrous oxide should be discontinued only after termination of operation. Analgesics should be administered to the child when he is still on the operating table.

## Anaesthesia with Ketamine, Nitrous Oxide, and Muscle Relaxants

Atropine is an obligatory component of premedication, while diazepam should be given for special indications. Initial and basal anaesthesia are induced by a single intravenous or intramuscular injection of ketamine with inhalation of nitrous oxide and oxygen in the 2:1 ratio. The main dose of the anaesthetic is 2-3 mg/kg for intravenous and 6-12 mg/kg for intramuscular injections, depending on the age of the child (the doses are the same as in multi-component anaesthesia with nitrous oxide without tracheal intubation). The trachea is intubated at the height of action of the main ketamine dose after intravenous injection of depolarizing muscle relaxants (depending on weight). The surgical stage of anaesthesia is maintained by repeated injections of  $\frac{1}{2}$  or  $\frac{1}{4}$  of the initial dose. Both depolarizing and non-depolarizing muscle relaxants can be used, depending on the extent of operative injury and the time of operation. The lungs are ventilated artificially during endotracheal anaesthesia either at a normal rate or with a moderate hyperventilation (with control of the acid-base balance), the nitrous oxide to oxygen ratio being 2:1. The last dose of the anaesthetic should be injected intravenously 15-20, and intramuscularly 40-50, minutes before the end of operation. Anaesthesia ends with inhalation of nitrous oxide and oxygen. When the wound is sutured, nitrous oxide supply should be discontinued and oxygen alone is used to ventilate the lungs till the moment of extubation. Adequate spontaneous respiration is usually restored by the end of operative intervention. The tracheal tube should be removed during the first minutes after spontaneous respiration is completely restored, because the laryngeal, pharyngeal and coughing reflexes are rapidly revived with this type of anaesthesia.

If necessary, the patient is decurarized by common doses of anticholinesterase preparations (proserine).

## Anaesthesia with Central Analgesics and Muscle Relaxants

*Promedol analgesic anaesthesia* Anaesthesia during operation is mainly maintained by promedol injections. Anaesthesia is induced by inhalation of nitrous oxide and oxygen (2:1) or by intravenous injections of sodium oxybate (100-120 mg/kg). When consciousness becomes confused,  $\frac{2}{3}$  of the initial promedol dose is injected intravenously. (The main dose of promedol is 3.5-4 mg/kg.) A narcotic sleep is induced in 15-20 seconds following the injection. Respiration becomes slow and deep. The respiratory function is usually inhibited (to complete cessation) in 30-40 seconds. Depolarizing muscle relaxants (2-2.5 mg/kg) are then injected intravenously. No muscular fibrillation is observed before tracheal intubation. The eyeballs are

fixed centrally, the pupils contract rapidly to the size of a point, and their response to light is flaccid, the corneal reflex vanishes. The skin is dry and warm, its colour is pale pink. The lungs are ventilated artificially with nitrous oxide and oxygen in the ratio of 2:1. The ventilation rate is either normal or slightly increased. Efficacy of lung ventilation is assessed by visual control and by determining the acid-base balance. Muscle relaxation during operation is maintained by fractional intravenous injections of tubarine in the corresponding doses.

If signs of insufficiently deep anaesthesia appear (tachycardia, motor reaction),  $\frac{1}{3}$  of the total promedol dose should be administered during the most dramatic stages of the operation. As a rule, this becomes necessary in 60-90 minutes after the initial administration of the analgesic. In the end of operation spontaneous respiration is inhibited and artificial lung ventilation with an air-oxygen mixture (2:1) should therefore be continued to complete restoration of adequate spontaneous respiration.

This type of anaesthesia is used in rare cases because of the danger of prolonged inhibition of respiration.

*Analgesic anaesthesia with promedol, nitrous oxide and halothane*  
Anaesthesia is induced by inhalation of nitrous oxide and oxygen (2:1) and halothane in a dose of 0.01-0.015 l/l (1-1.5 per cent v/v). After the patient is asleep, promedol is injected intravenously  $\frac{2}{3}$  of total dose (the total dose is 1-1.5 mg/kg). The trachea is intubated after relaxation of the muscles (with depolarizing relaxants). Relaxation during operation is attained by tubarine (0.3 mg/kg). The lungs are ventilated during anaesthesia with a mixture of nitrous oxide and oxygen (2:1) and halothane in a dose of 0.004-0.005 l/l (0.4-0.5 per cent v/v). The remaining  $\frac{1}{3}$  of the total promedol dose is injected intravenously in 50-60 minutes after the operation is started if clinical signs of insufficient anaesthesia appear. First signs of recovering senses appear in children in 5-7 minutes after the anaesthetic inhalation is discontinued (the child reacts to the tracheal tube). As soon as the artificial lung ventilation is stopped, spontaneous respiration is restored.

*Nitrous oxide and pentazocine anaesthesia*  
Atropine and diazepam are obligatory components of premedication. Anaesthesia is induced by inhalation of nitrous oxide and oxygen in the ratio of 2:1 (for 5-7 minutes). Diazepam is injected intravenously in a dose of 0.3-0.5 mg/kg, pentazocine is injected intravenously, immediately after diazepam, in a dose of 1.2-3 mg/kg, depending on age (the dose is the same as in multi-component anaesthesia with nitrous oxide without muscle relaxants). A pentazocine injection ensures an adequate analgesic effect for 40-50 minutes. The maximum effect of the main analgesic dose is attained in 2-3 minutes. Depolarizing muscle relaxants are then administered and the trachea intubated. The lungs



are ventilated artificially with nitrous oxide-oxygen mixture (2:1). Relaxation during operation is maintained by fractional administrations of depolarizing or non-depolarizing muscle relaxants. If clinical signs of insufficient anaesthesia develop in 40-50 minutes after the first pentazocine administration,  $\frac{1}{2}$  or  $\frac{1}{4}$  of the main pentazocine dose (depending on anticipated length of operation) should be injected to maintain anaesthesia. The last dose of the analgesic should be administered 40-50 minutes before the end of operation. Inhalation of nitrous oxide and oxygen continues till the moment when skin sutures are placed, after which the supply of nitrous oxide is discontinued. The endotracheal tube is removed during the first minutes after restoration of adequate spontaneous respiration.

### Anaesthesia with Neuroleptanalgesia

Atropine and thalamonal are commonly used for premedication in neuroleptanalgesia (1 ml of thalamonal contains 2.5 mg droperidol and 0.05 mg phentanyll). The dose of thalamonal depends on the body weight of a child: 0.5-1 ml for a child weighing from 10 to 20 kg, 1-1.5 ml for a child of 21-40 kg, and 1.5-2 ml for children weighing 41-50 kg. The preparations are usually administered intramuscularly, 40-50 minutes before operation. Thalamonal has a marked sedative effect in emotionally labile children. Induction begins with inhalation of nitrous oxide and oxygen in the ratio of 2:1. Light sleep and analgesia occur in a few minutes provided the premedication was adequate. During this period the vein is punctured and catheterized and droperidol is administered in the following doses: 3-4 ml (7.5-10 mg) to children weighing 10-20 kg, 4-6 ml (10-15 mg) to children of 21-40 kg, and 6-8 ml (15-20 mg) to children weighing 41-50 kg. Slow injection of droperidol ensures a moderate fall in the arterial pressure and accelerates the pulse rate during induction anaesthesia. The effect of the preparation is seen in 5 minutes. It attains its maximum in 20 minutes and lasts for 2-3 hours. Droperidol induces sleepiness, full emotional estrangement and indifference. The child shows no signs of fear or anxiety. His movements and speech are slow, the coordination is upset. Spontaneous respiration is not practically affected.

Intravenous administration of phentanyll (2-3 minutes after droperidol injection) in a dose of 0.008-0.01 mg/kg deepens the described symptoms. The respiration is inhibited to complete apnoea accompanied with convulsive rigidity of the trunk muscles. Artificial lung ventilation should be begun in 20-30 seconds after the administration of the analgesic. Depolarizing muscle relaxants are given as in other cases, and then the trachea is intubated. The patient falls asleep after inhalation of the nitrous oxide-oxygen mixture (2:1). The muscles are relaxed by administration of depolarizing or non-

depolarizing muscle relaxants. The lungs should be ventilated artificially during anaesthesia.

Clinical signs of analgesic insufficiency, such as accelerated heart rate and a slightly elevated arterial pressure, are indications for repeated injections of phentanyl (first in 20-30 and then in 40-60 minutes). Analgesia is maintained by administering  $\frac{2}{3}$  or  $\frac{1}{3}$  of the initial dose of phentanyl. The last dose of the analgesic should be administered 20-30 minutes before the end of operation. The droperidol effect lasts for 2-3 hours, no additional doses are therefore required if the operation ends before expiration of this time. If the operation lasts longer,  $\frac{2}{3}$  of initial droperidol dose should be administered in 90-120 minutes. If common doses are used with children, they wake up at the end of operation as soon as the supply of nitrous oxide is discontinued. The operated children are usually quiet, sleepy and present no complaints. This condition lasts for 6-8 hours.

## Chapter 11

### Artificial Hypothermia

Hypothermia is a pathological condition, but since the oxygen demands of the body decrease and the resistance to oxygen deficit increases in this condition, hypothermia is used in clinical practice.

*Physiological grounds.* Artificial hypothermia prevents irreversible changes in the central nervous system during oxygen deficit. The fall of body temperature to 30°C decreases oxygen demand by almost 50 per cent. Hypothermia thus increases body resistance to hypoxia by 100 per cent. Artificial hypothermia can be slight, moderate and deep. In slight hypothermia the body temperature decreases to 34°C, in moderate to 28°C, and in deep hypothermia to 8-12°C. The deeper hypothermia, the longer the brain can remain unchanged in conditions of the absence of oxygen. When conducting hypothermia, the reaction of the human body to cold should be prevented artificially. Some methods have been proposed for this purpose: 1—deep anaesthesia (stage III<sub>3</sub>). It is not however recommended to use this method because deep anaesthesia has an adverse effect on the body, 2—deep neuroplegia. This method is not practically used now because neuroplegics inhibit the adaptation and compensatory mechanisms of the body, which are necessary during operation and anaesthesia, 3—combination of shallow anaesthesia (stage III<sub>1</sub>) with deep curarization. This is the most popular method. Hypothermia in conditions of shallow anaesthesia and relaxation of muscles with non-depolarizing agents is the most advantageous because of its indisputable advantages over the above-mentioned methods.

*Indications* Hypothermia in children is indicated for the following operations 1—operations on large vessels whose complete or partial occlusion can upset cerebral circulation for more than three minutes, 2—operations on the heart continuing not longer than 5-7 minutes, 3—prolonged operations on the heart with extracorporeal circulation of blood

Hypothermia is indicated as a therapeutic means in a—hyperpyrexia, b—severe injury of the brain accompanied by hypoxia, c—complex treatment of post-hypoxic brain affections, e.g. oedema

*Procedure* The body temperature can be safely decreased only in an anaesthetized child. Multi-component shallow endotracheal anaesthesia with non-depolarizing muscle relaxants should be used. Depolarizing muscle relaxants can be used with 2-3 year-old children. The temperature in the oesophagus should constantly be determined during hypothermia, ECG and EEG should also be permanently taken.

Two methods are used to attain hypothermia by cooling the body surface, and by cooling the patient's blood outside the body (with extracorporeal circulation). Surface cooling is achieved by immersing the child in cold water (8-10°C), placing ice-bags on his body, blowing cold air (by fans or in special chambers), or by packing the child in a special blanket inside which a coolant is circulated.

Patient's blood is cooled outside the body in a special heat exchanger included in the apparatus for extracorporeal blood circulation. Deep hypothermia can be attained only with this latter method, because the heart's action is arrested at temperatures below 20°C.

After operation the child should be warmed up in water at a temperature of 38-42°C. When the temperature in the mouth rises to 35°C, the supply of anaesthetics should be discontinued. The child shows signs of recovering senses. After cleaning the tracheobronchial tree the endotracheal tube is removed and the lungs are ventilated with oxygen through the mask of the anaesthetic apparatus until adequate spontaneous respiration is completely restored.

## Chapter 12

### Local Anaesthesia

Desensitization of tissues in the operating field using physical (cooling, compression) or chemical agents is called local anaesthesia. At present physical methods of desensitization are no longer used, while chemical substances are widely employed in modern practice. Local anaesthesia alone is only of relative importance in paediatrics because the child remains conscious during operation, and this is undesirable. But various types of local anaesthesia are widely used.

as components of general anaesthesia, or independently as analgesic means during the post-operative period

The advantages of local anaesthesia are as follows 1—reliable blockade of pain impulses from the operating field, 2—minimum effect on the organs and systems of the patient, 3—easy availability in any conditions, application without any expensive tools or apparatuses

Local anaesthesia 'per se' has disadvantages too The most significant of them are the following a—the patient remains conscious and a psychic injury is thus possible, operations with local anaesthesia are very difficult with most children, b—absence of control of vital body functions, local anaesthesia is practically inapplicable to cases where operations should be performed on the thoracic organs, neuro-surgical operations and intervention on the abdominal organs are also quite restricted, c—serious complications are possible

Substances blocking perception or transmission of pain are called local anaesthetics Procaine, trimecaine, xycaïne, and dicaine are commonly used for local anaesthesia in children The doses are specified in Table 17

Table 17 Doses and Concentrations of Preparations Used for Local Anaesthesia in Children

| Preparation                    | Concentration of solution, in per cent | Permissible dose, as dry substance, mg/kg           | Administration                          |
|--------------------------------|--|---|---|
| Procaine                       | 0.25-0.5                               | 20  | for infiltration anaesthesia            |
|                                | 5-10                                   |   | for surface anaesthesia                 |
| Xycaïne (xylocaine, lidocaine) | 0.25-0.5                               | 15  | for infiltration anaesthesia            |
|                                | 1-3                                    |   | for conduction and epidural anaesthesia |
|                                | 5                                      |   | for surface anaesthesia                 |
| Trimecaine                     | 1-3                                    | 15  | for conduction and epidural anaesthesia |
| Dicaine                        | 0.5-1                                  | 1 (0-1 year)<br>2.5 (1-6 years)<br>3-6 (6-12 years) | for surface anaesthesia                 |

**Terminal (surface) anaesthesia** Terminal anaesthesia is the simplest and long-known method of desensitization of mucosa Anaesthesia is attained by direct contact of the anaesthetic solution with the mucosa (administered as drops, by aeration, application of tampons soaked in anaesthetic solution, etc.) Terminal anaesthesia is widely used in paediatric ophthalmology for anaesthesia of the conjunctival sac It is also used for treatment of diseases of the ear, nose and throat, and in thoracic surgery It may be suitable for direct laryngoscopy, bronchoscopy, and the like manipulations in nurslings and in se-

rior schoolchildren Surface anaesthesia should be used as an additional means to suppress undesirable reflexes and to prevent complications For example, a 5 per cent procaine solution is filled into the tracheobronchial tree to prevent bronchospasm and to inhibit the coughing reflex during bronchoscopy The vomiting reflex is inhibited by applying anaesthetic to the mouth mucosa (with tampons soaked in the anaesthetic solution) Terminal analgesia of the urethra is obligatory for endoscopy of boys (even in conditions of anaesthesia) It facilitates the passage of the tool through the urethra and spares the child of unpleasant sensations associated with minor injuries to the urethra

Terminal analgesia is attained by 0.5-1 per cent solutions of dicaine, 5-10 per cent solutions of procaine, and 3-5 per cent solutions of trimecaine and xycaïne The anaesthetics are readily absorbed by the mucosa and this should be remembered in order to prevent overdosage and poisoning

**Infiltration anaesthesia.** This consists in layered infiltration of soft tissues in the field of operation using weak anaesthetic solutions The method of creeping infiltration has been worked out in detail by the Soviet physician A. Vishnevsky He also proposed the following composition of a 0.25 per cent solution of the anaesthetic: procaine 2.5 g, adrenaline (1:1000) 2 ml, sodium chloride 5 g, potassium chloride 0.075 g, calcium chloride 0.125 g, and distilled water to make 1000 ml The solution has a good analgesic effect and its ionic composition is close to that of the interstitial fluid At present a 0.25 per cent trimecaine solution for infiltration anaesthesia is prepared in the same way

This type of anaesthesia is used in minor operations (herniotomy, cordectomy, etc.) It is used for removal of small newgrowths of soft tissues (of the angioma type) in neonates and infants When carrying out infiltration anaesthesia, it should be remembered that tissues are very thin in children It is more correct to anaesthetize each successive layer with visual control Infiltration anaesthesia is more frequently used for additional anaesthesia of reflexogenic zones in vast surgical operations on the organs of the chest, abdomen and the pelvis For example, for anaesthesia of the lung root, mediastinum, mesenteric radix, pelvic plexus, etc This decreases the flux of pathological impulses to the central nervous system and stabilizes blood circulation of a child during vast surgical operations

**Regional anaesthesia** This type of anaesthesia includes intravenous, conduction, paravertebral, epidural, and cerebrospinal anaesthesia All types of regional anaesthesia are characterized by the following 1—the anaesthetic solution is administered not directly into the operative zone but at a distance from it, topography of the operative field is not therefore disturbed, 2—relatively small amounts of anaesthetics are used, while anaesthesia and relaxation of

muscles are attained over a large area of the body, 3—good knowledge of anatomy and anaesthetic techniques is necessary, otherwise efficacy of anaesthesia cannot be guaranteed and complications are more likely to occur

Regional anaesthesia in children can be used 'per se' or in combination with general anaesthesia, as well as in post-operative analgesia

*Intravenous anaesthesia* The method was first proposed by Bier in 1908 The method for using this anaesthesia in children was elaborated in detail by the Soviet physician Isakov (1960) It is used independently in senior children for surgery of the extremities, but it can also be used in children of any age in combination with premedication and nitrous oxide-oxygen anaesthesia The technique is simple A cuff of a tonometer is placed on the extremity (above the site of operation) dehaematized preliminarily by an elastic bandage or by lifting the extremity A 0.5 per cent procaine or trimecaine solution is administered into a peripheral vein in doses specified in Table 18

Table 18 Doses of 0.5% Procaine Solution (in ml) for Intravenous Anaesthesia of Children

| Age, years | Upper extremity |         |       | Lower extremity <sup>1</sup> |       |       |
|------------|-----------------|---------|-------|------------------------------|-------|-------|
|            | shoulder        | forearm | hand  | thigh                        | shin  | foot  |
| 1-3        | 40-50           | 20-30   | 15-20 | 50-60                        | 40-50 | 20-30 |
| 4-6        | 50-60           | 30-40   | 20-30 | 60-70                        | 50-60 | 30-40 |
| 7-10       | 60-70           | 40-50   | 20-30 | 70-80                        | 60-70 | 40-50 |
| 11-15      | 70-80           | 50-60   | 30-40 | 80-100                       | 70-80 | 50-60 |

The effect of anaesthesia is adequate in 8-10 minutes and it lasts until the cuff is removed The pressure in the cuff is slowly decreased at the end of operation Sensibility is restored in 5-6 minutes after removal of the cuff Two cuffs ensure a better dehaematization one is placed above and the other below the operating field and the anaesthetic is administered in the site between them

The disadvantages of the method are the following 1—the time of operation is limited pain occurs if the cuff remains on the limb for a long time, 2—the 'vein problem' in infants, 3—absence of anaesthesia of the operated limb after operation 4—possible passage of the solution outside the zone limited by the cuff and possible intoxication

*Anaesthesia into haematoma* A 1.5 per cent trimecaine or xycaïne solution is injected into the haematoma in the zone of bone fracture Anaesthesia develops in 6-10 minutes and lasts for the time suffi-

cient for reposition of the bone fragments. General anaesthesia is usually impossible with children delivered in an ambulance car (accidental bone fractures) because their stomachs are 'full'. Anaesthesia into the zone of fracture is therefore more reasonable in such cases. The needle is passed into the injured site and the piston pulled back slightly until blood appears in the barrel. Then 10-20 ml of tetracaine solution are injected.

The disadvantage of this method is the absence of muscular relaxation in the zone of fracture. This type of anaesthesia is mainly used in fractures of the forearm and shin bones.

*Conduction anaesthesia* This method is used to anaesthetize nerve trunks and plexuses. A small amount of anaesthetic solution is injected in the region of the nerve trunk and plexus to interrupt the transmission of impulses and to ensure anaesthesia and relaxation of muscles in the entire innervated zone. Conduction anaesthesia is therefore considered to be the most effective method which is being constantly improved and seems to be quite promising. The brachial plexus, sciatic and femoral nerves are usually anaesthetized by this method. Conduction anaesthesia in paediatrics is mainly used for operations on the extremities in combination with nitrous oxide-oxygen anaesthesia. It may be used independently in senior children. It is advantageously used for post-operative analgesia and for therapeutic blockade.

Until recently the main difficulty in conduction anaesthesia in children was accurate delivery of the anaesthetic solution to the nerve trunk or plexus. Even good knowledge of anatomy and projection points of nerve trunks on the skin cannot guarantee successful anaesthesia because of the high variability of the nerve trunk position in human individuals. The situation has markedly changed after development of the method for electric excitation of nerve trunks, which not only guarantees adequate anaesthesia but facilitates substantially the location of the nerves. Consider, by way of illustration, anaesthesia of the brachial plexus in children (Fig. 26). The child is anaesthetized by inhalation of nitrous oxide and oxygen and the needle is inserted into the armpit perpendicularly to the shoulder bone (using the brachial artery as the landmark). As the needle passes through the tissues, a weak current (0.3-0.6 mA) is applied. When the needle reaches the nerve trunk of the brachial plexus, the corresponding muscles of the forearm and hand contract in response. This is the indication for injection of the anaesthetic solution. Tetracaine (1-1.5 per cent solution) or xylocaine with adrenaline (1:200,000) is used in the dose of 10-15 mg/kg. The anaesthetic effect is attained in 7-10 minutes and extends over the middle portion of the shoulder to last for 3-4 hours. Hypoesthesia of the extremity lasts for another 6-10 hours to solve the problem of post-operative analgesia. The femoral and sciatic nerves are anaesthetized in a similar way.

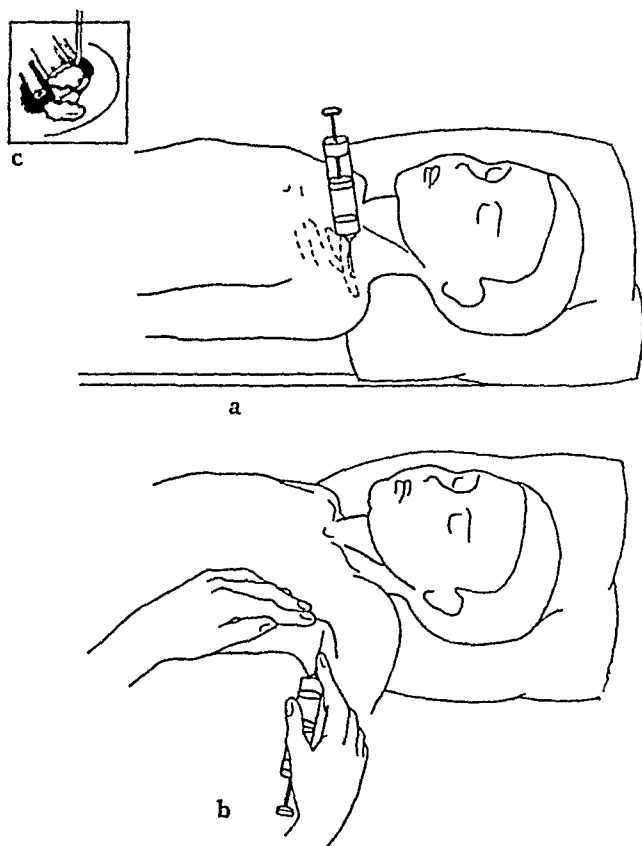


Fig 26 Conduction anaesthesia of brachial plexus

*a*—supraclavicular approach, *b*—axillary approach, *c* —irrigation of nerve trunks

Blood vessels usually run along the nerve trunks and therefore, in order to prevent their puncture, the piston should be slightly pulled back to see that no blood enters the syringe. The puncture of the blood vessel is safe in itself and some authors do not regard it as a complication, but entrance of the anaesthetic into the circulatory system causes poisoning. Sharp needles should be used to prevent injury to the nerve.

**Paravertebral anaesthesia** Desensitizing intercostal and lumbar nerves at their exit from the intervertebral foramina is called paravertebral anaesthesia. It is necessary to determine correctly the zone of anaesthesia and to block the two segments above the region innervated by the nerve of the operative region. Each segment is anaesthetized separately by injecting 4-6 ml of a 0.5 per cent trimecaine or lidocaine solution into one site (without exceeding the permissible age dose). The method is complicated and short-acting and it is



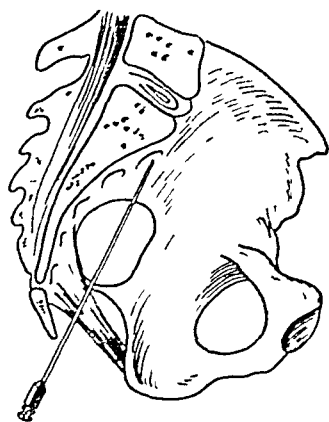


Fig 27 Presacral anaesthesia

therefore rarely used in children. It can be used for analgesia in injuries and after operations on the abdominal and thoracic organs

*Parasacral anaesthesia* is actually a modification of paravertebral method. It consists in blockade of nerves at their exit from the sacral hiatuses. It ensures adequate anaesthesia of the pelvic organs and is used in paediatric urology and proctology in combination with surface anaesthesia. The approach to the nerves is between the rectum

and the anterior surface of the sacrum. The needle should slide over the bone in the direction of the hiatuses (Fig 27). A 0.5 per cent solution of trimecaine, lidocaine, or procaine are used for anaesthesia. The effect is attained in 10-15 minutes after the injection. The danger of puncturing the rectum should be considered in a parasacral anaesthesia, the cellular tissue of the small pelvis can also be infected.

*Peridural (epidural) and caudal (sacral) anaesthesia* These types of anaesthesia include the administration of anaesthetic solutions into the epidural space, which is found between the dura mater and the interior surface of the cerebrospinal canal. The epidural space in children is larger in the lumbar region (6 mm), while it does not exceed 2-4 mm over the remaining length. The epidural space contains many venous plexuses, loose connective tissue and fat. Anterior and posterior radices of the cerebrospinal nerves pass through this space.

Nowadays in connection with development of pharmacology of anaesthetics and improvement of techniques, epidural anaesthesia is widely used as a component of or an independent anaesthesia in operations on the abdominal organs, pelvic organs, and the urinary

Table 19 Site for Epidural Puncture Depending on the Operation Field

| Operation field                | Site of puncture                  |
|--------------------------------|-----------------------------------|
| Chest                          | T <sub>III</sub> -T <sub>V</sub>  |
| Epigastrium                    | T <sub>VI</sub> -T <sub>IX</sub>  |
| Mesogastrium and hypogastrium  | T <sub>VIII</sub> -L <sub>I</sub> |
| Pelvic organs                  | T <sub>X</sub> -L <sub>II</sub>   |
| Perineum and lower extremities | L <sub>II</sub> -L <sub>IV</sub>  |

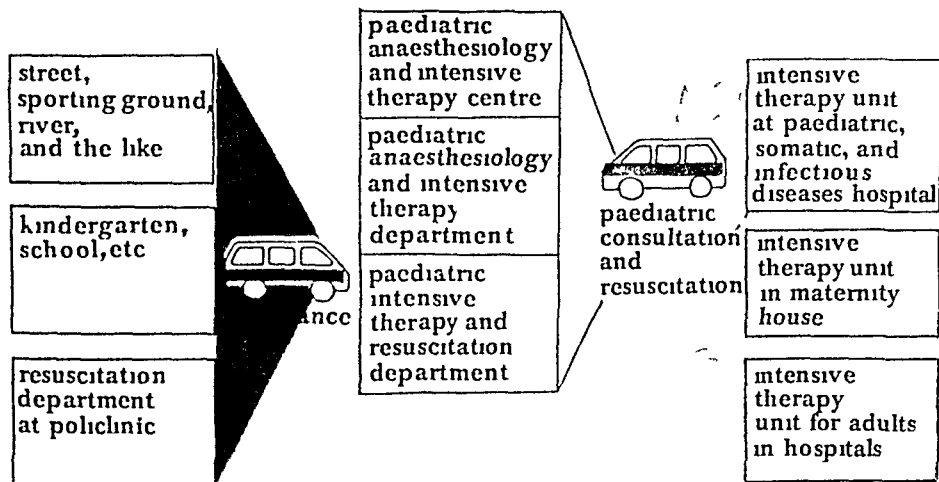


Plate 1 Resuscitation service in a large city

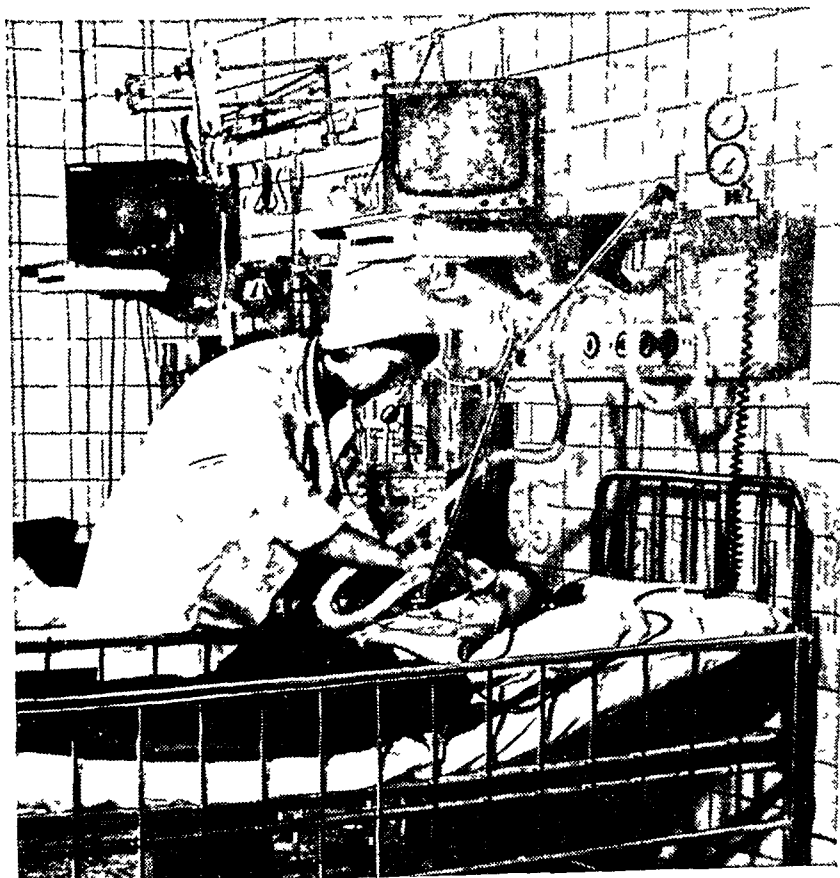
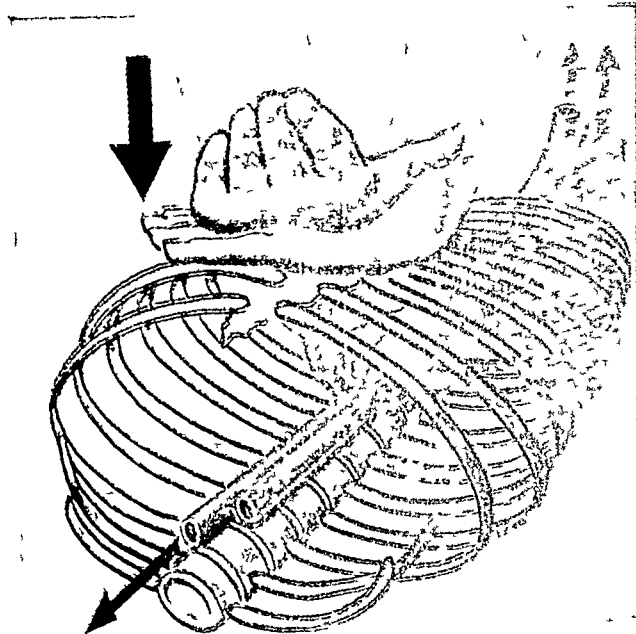
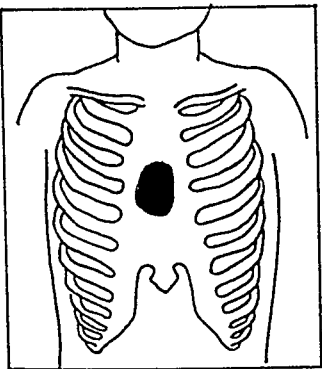
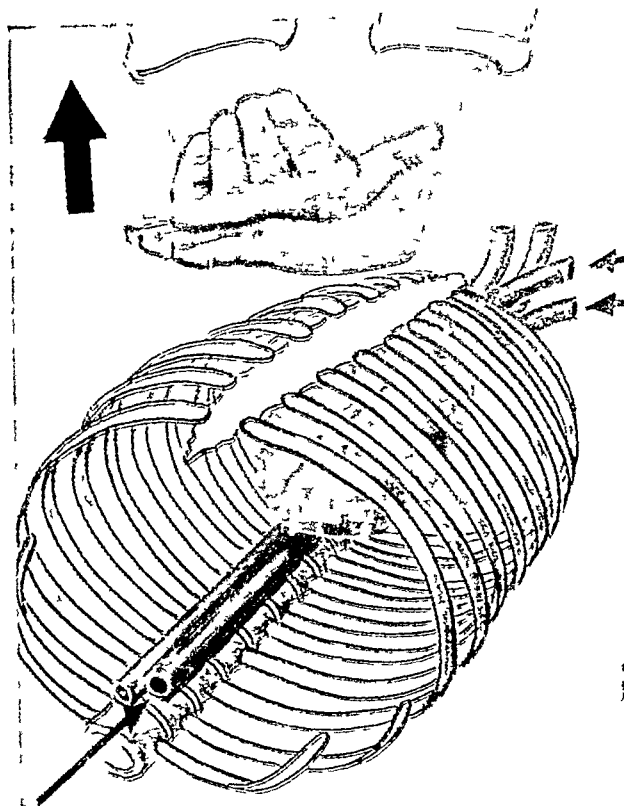


Plate 2 Paediatric intensive therapy unit



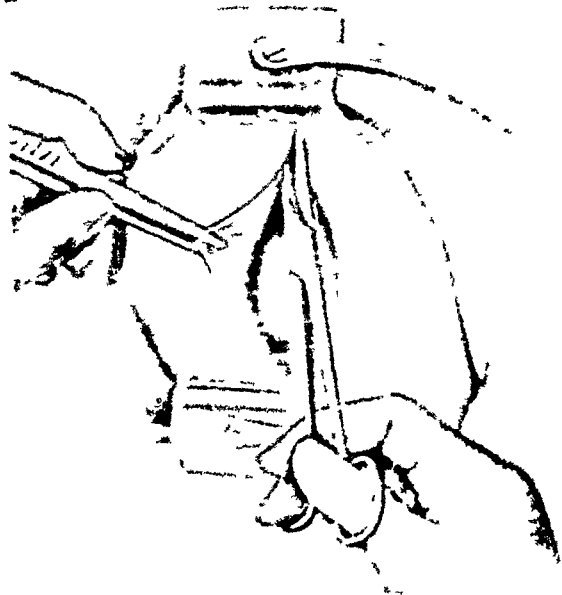


*a*



*b*

Plate 4 Closed chest cardiac massage  
 (a) artificial systole the heart is compressed between the sternum and the vertebral column,  
 (b) artificial diastole blood is admitted to the heart

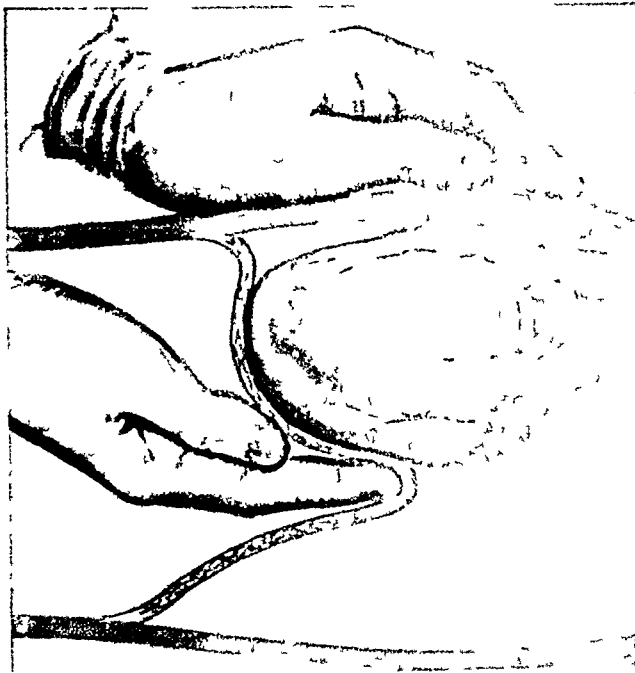


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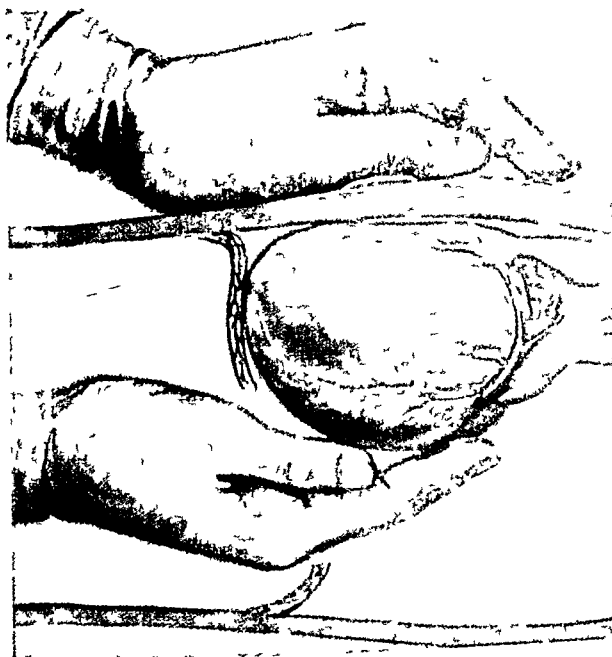


c





*a*



*b*

Plate 6 Open chest cardiac massage (transdiaphragmatic access)  
 (a) through intact diaphragm,  
 (b) through incised diaphragm

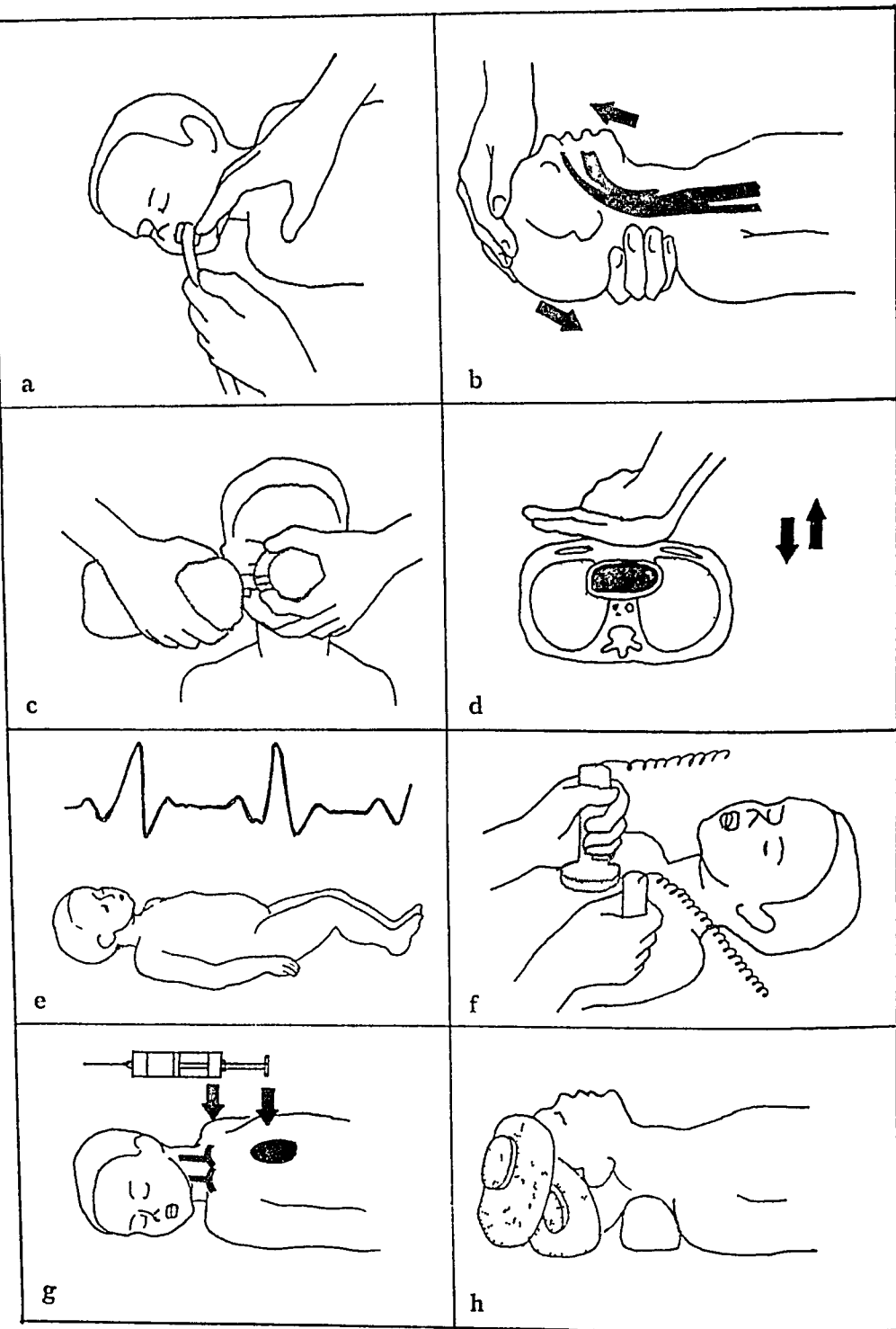


Plate 7 Resuscitation measures

(a) cleaning airways,  
 (b) deflexing the head,  
 (c) giving artificial ventilation,  
 (d) closed chest cardiac massage,  
 (e) taking ECG,

(f) applying defibrillator,  
 (g) injections, calcium chloride,  
 adrenaline, sodium bicarbonate,  
 (h) applying cold

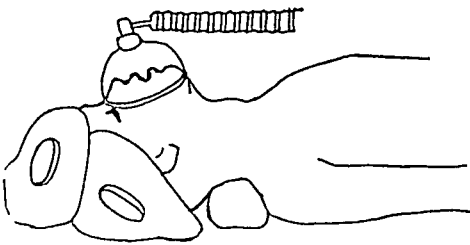
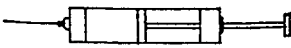
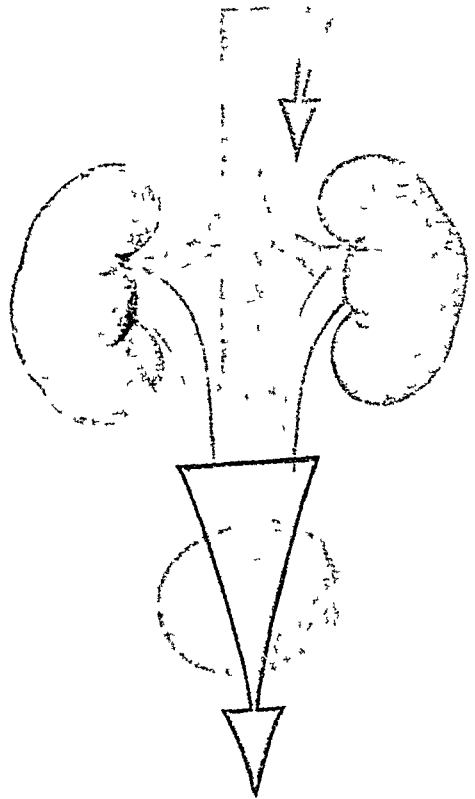
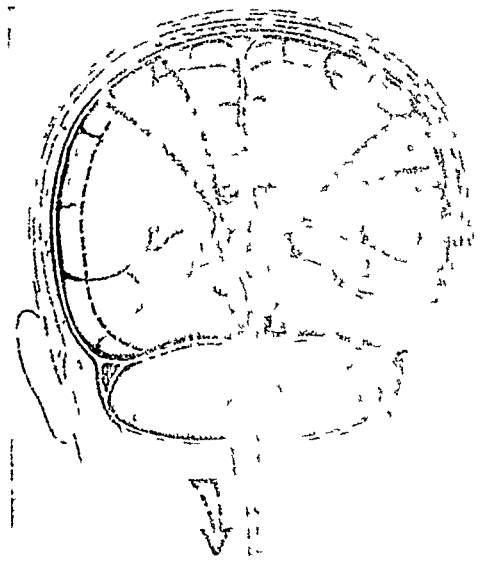


Plate 8 Intensive therapy of brain oedema by dehydration, correcting homeostasis, applying cold, and administering hormones



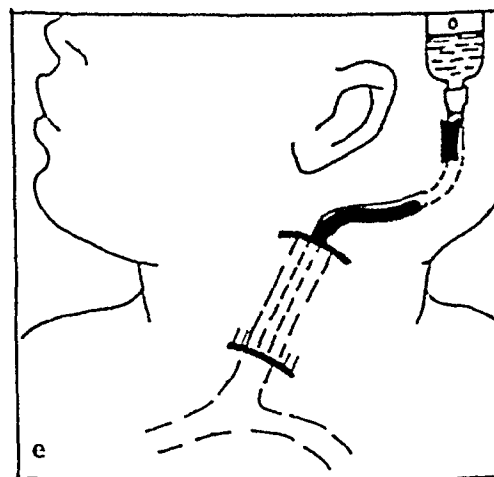
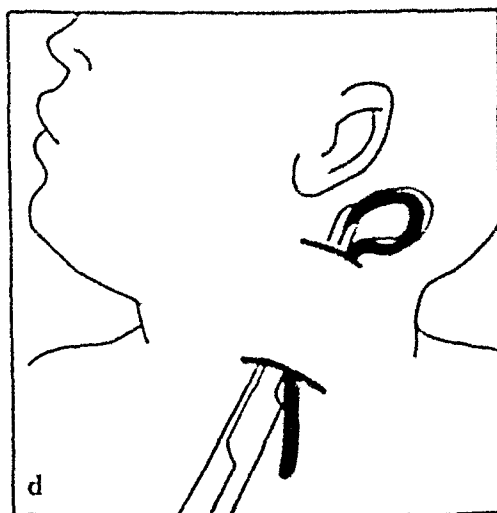
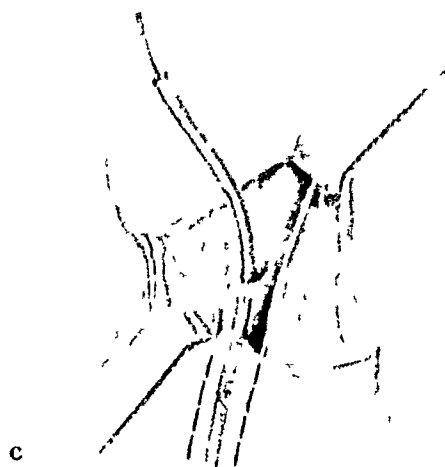
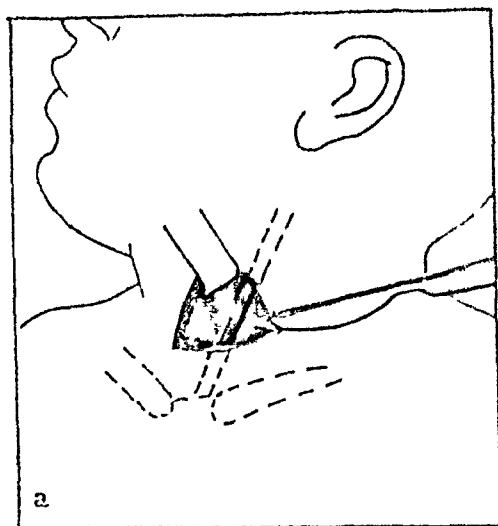


Plate 9 Open cannulations of the external jugular vein

- (a) skin incision and separation of the vein,
- (b) puncturing the vein and introducing the conductor,
- (c) passing a catheter by the conductor,
- (d) forming a skin tunnel and passing the catheter end through it,
- (e) fixation of the catheter and connection to the system

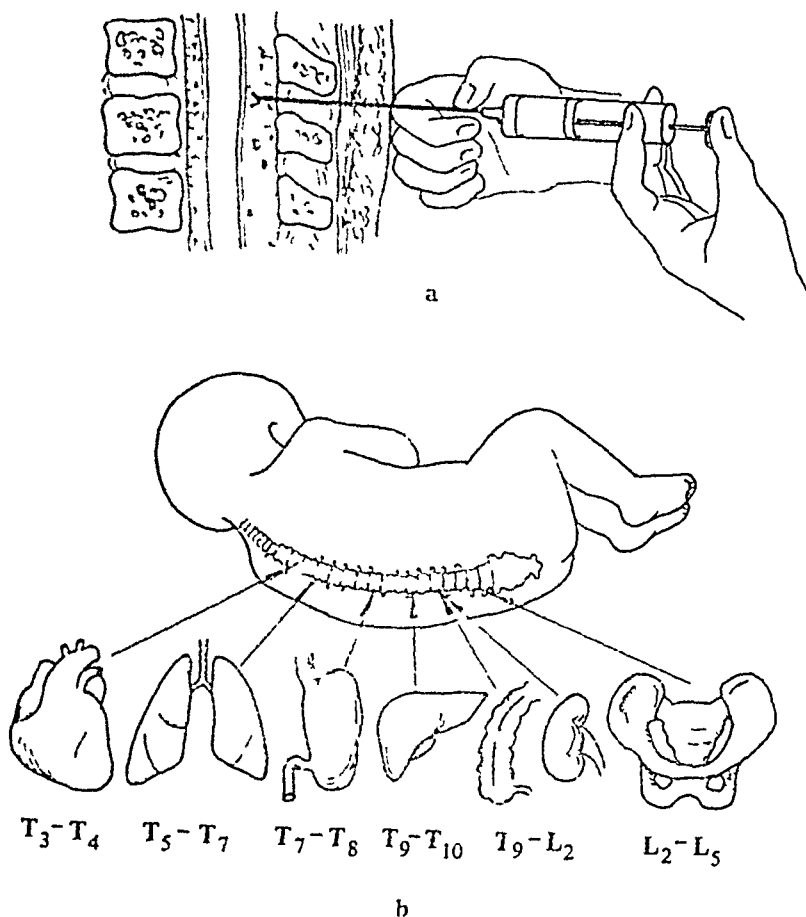


Fig 28 Epidural anaesthesia

*a*—puncture of epidural space, *b*—sites of injections for various operations

system Epidural anaesthesia is a common method of prolonged post-operative analgesia. The child is placed on his side, his thighs are flexed on the abdomen (Fig 28). The site of puncture depends on the required level of anaesthesia (Table 19).

The manipulation is usually conducted with local infiltration anaesthesia but infants and children may be given anaesthesia with nitrous oxide and halothane, especially so if multi-component analgesia will be further used. A needle with a rounded end is passed strictly medially through the interspinal and yellow ligaments. The operator feels as if the needle enters an empty cavity when it reaches the epidural space. A 2-ml syringe is now attached to the needle. If the needle's position is correct, an isotonic sodium chloride solution freely passes into the epidural space. It is necessary to check if the needle is in the vein or the epidural space by pulling back the sy-

ring piston if the needle is in the center of the ring; and if the needle is in the center of the ring, it will pierce the ring (Dogliotti's test). If the needle is in the center of the ring, it will pierce the ring (Dogliotti's test). If the needle is in the center of the ring, it will pierce the ring (Dogliotti's test).

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The analgesic effect during operation was evaluated by the visual estimation of morphine in a dose of 0.1-0.2 mg (1-2 drops of 1% solution) 1-2-4-6 hours), promedol in a dose of 0.2-0.3 mg (2-3 drops of 0.1% solution) 2-3 hours), and phentonyl in a dose of 0.01-0.02 g (1-2 drops of 0.1% injections in 60-90 minutes).

During the post-operative period for the same patients administered are the following morphine, 0.05-0.2 mg/kg (the effect lasts for 48 hours), promedol, 0.1-0.15 mg/kg (the effect lasts to 12 hours) and phentanyl, 0.002-0.005 mg/kg (the effect lasts to 2-3 hours).

Epidural anaesthesia with surface narcosis makes it possible to carry out operations in children with adequate spontaneous respiration and stable blood circulation. This anaesthesia ensures complete analgesia during the post-operative period, improves the external respiration function, the intestinal function and normalizes peripheral blood circulation. In order to prevent infection during prolonged epidural anaesthesia, penicillins are added to the anaesthetic solution.

As distinct from cerebrospinal anaesthesia, epidural anaesthesia does not cause headache, slight hypotension occurs infrequently. Respiratory distress is rare too. Complications occur only if the anaesthetic is injected into the cerebrospinal fluid due to occasional puncture of dura mater or catheterization of the vein in the epidural space.

(when a large dose of the preparation enters the blood circulation system)

Caudal anaesthesia does not differ substantially from epidural anaesthesia. The analgesic effect is attained by administering the anaesthetic into the distal part of the epidural space through the sacral hiatus. The solution reaches  $L_1$  and is distributed in the entire region innervated by the sacrolumbar plexus (Fig 29). The spinal cord

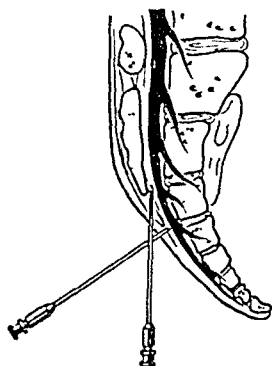


Fig 29 Caudal anaesthesia

extends from the atlas to the second lumbar vertebra, less frequently to the second sacral vertebra. At this level the distal part of the spinal cord forms the medullary cone, which extends to the long terminal filament. The epidural space continues to the sacrolumbar ligament, which covers the sacral hiatus. These anatomical features account for the safety and reliability of the sacral anaesthesia, which ensures adequate anaesthesia of the organs of the small pelvis, the perineum, and the lower extremities.

The procedure is very simple: the child is placed face down, with the pelvis slightly elevated, or on his side. The needle, with the mandrin inside it, is passed through the membrane between the sacral horns at an angle of  $45^\circ$  until it meets the bone; its position is then changed to horizontal and the needle is moved further through 2-3 cm parallel to the sacrum. After an aspiration test for correct position of the needle, 1.5-3 per cent solution of trimecaine or lidocaine with adrenaline (1:200,000) is injected, or a plastic catheter is passed for prolonged caudal anaesthesia.

Caudal anaesthesia is used in paediatrics together with shallow nitrous oxide-oxygen narcosis during operations on the perineum and the lower extremities. Independently it is used for post-operative analgesia. In addition to the analgesic effect, epidural anaesthesia causes some other effects, e.g. sympathetic blockade. The cholinergic effect is intensified and the intestinal peristalsis is increased by this anaesthesia.

#### COMPLICATIONS OF LOCAL ANAESTHESIA, THEIR PREVENTION AND TREATMENT

Local and general complications associated with local anaesthesia are distinguished. General complications are poisoning with anaesthetics and anaphylactic reactions. Poisoning with anaesthetics can occur 1—during occasional puncture of the blood vessel

and injection of anaesthetics into the blood circulation system; 2—as a result of puncture of dura mater when the dose of anaesthetics, intended for epidural injection, is injected into the cerebrospinal canal, 3—as a result of overdosage

Poisoning is prevented by regularly checking if the anaesthetic is not injected into the blood vessel, cerebrospinal canal, by strictly observing the prescribed doses, and by adding adrenaline to the anaesthetic solution to decrease its absorption

Signs of poisoning differ by their intensity. Slight forms of poisoning are manifested by nausea, vomiting, flaccidity, or excitation. Generalized clonic convulsions with respiratory distress (to complete cessation of respiration) are characteristic of grave poisoning.

In the presence of signs of slight poisoning, the administration of the anaesthetic should immediately be discontinued and additional amounts of liquid should be administered intravenously or much liquid should be given to drink. In severe poisoning assisted or artificial respiration should be given with narcotization to stage III<sub>1</sub>–III<sub>2</sub> to decrease excitation and to relieve convulsions. Hexenal is usually used for the purpose, but if an intravenous injection is difficult, nitrous oxide and halothane should be given without delay. Glucose with insulin (20–25 per cent of the daily volume of liquid taken) should then be infused in order to accelerate withdrawal of excess anaesthetic from the body.

Cases of hypersensitivity to anaesthetics are not infrequent. But this may be prevented by studying appropriately the anamnesis of each patient. The manifestations of hypersensitivity to anaesthetics are different. They may vary from allergic reactions, such as nettle rash, itching or local oedema of subcutaneous fat, to severe anaphylactic shock. The anaesthetic administration should be suspended immediately in any case. If the response is moderate, the child can be given calcium chloride, diazolin, diphenhydramine hydrochloride, pipolphen, and small doses of promedol. Severe cases should be treated by energetic infusion of adrenaline, hydrocortisone and pipolphen, assisted respiration by mask of the anaesthesia apparatus or a simple ventilator is sometimes necessary.

Local complications include injuries of nerve trunks, vessels (with haematomas), infection, and inflammation of soft tissues. These complications can be prevented by carrying out appropriately the anaesthetic operations, including a proper selection of needles and strict observation of aseptic conditions.

## Chapter 13

### Dependence of Anaesthesia on the Child's Condition and Character of Operation

The special character of anaesthesia depends mainly on the child's condition and the character of surgical operation, as well as the age of the child

**Minor operations in out-patient conditions** Anaesthesia in minor operations is as important as narcotization of patients for any other surgical operation. The term minor does not apply to anaesthesia, because severe complications can occur even in short-lasting narcosis.

*Preparing* the patient for anaesthesia includes emptying his stomach through a gastric tube (if he ate for the last time 3-4 hours before anaesthesia). A hospitalized patient should be premedicated as usual. If the operation is to be made in out-patient conditions, atropine should be administered, or no premedication is required at all.

The patient should be *anaesthetized* with nitrous oxide and oxygen, or nitrous oxide, halothane and oxygen, propanidid or barbiturates should be administered intravenously, older children require local anaesthesia.

**Emergency operations** Whenever necessary to correct the water-salt balance and blood circulation, indicated is intravenous administration of blood, plasma, polyglucin, 10 per cent glucose solution, and electrolytes. If a child ate within 3-4 hours before operation and in the presence of acute abdomen, it is necessary to empty his stomach through the tube that should remain in the stomach during the entire operation.

*Anaesthesia* should be given through an endotracheal tube or by mask inhalation. Intravenous anaesthesia is indicated for older children during short-lasting operations.

**Surface operations on the head** Anaesthesia should be given either by mask or endotracheally, depending on the site of surgical operation. Local anaesthesia is possible with older children.

**Craniotomy and operations on the brain** These operations are usually conducted with endotracheal anaesthesia. Adequate premedication is necessary to prevent excitation. Reinforced endotracheal tube should be used; it can be bent at any desired angle.

**Operations for correction of cleft lip and palate and for Pierre Robin's syndrome** These operations are performed with endotracheal anaesthesia. The tube is usually passed through the mouth, but in some cases it is more convenient to introduce it through the nose. Intubation may be difficult. Tamponade of the pharynx is necessary to prevent aspiration of blood. Replenishment of blood loss is necessary. Anaesthesia should be done with nitrous oxide and halothane, and also with oxygen.

**Operations on the neck.** These include opening of small abscesses, extirpation of cysts with anaesthesia administered through the mask, deep phlegmonas and submandibular abscesses should be opened with endotracheal anaesthesia

**Tracheotomy.** This should preferably be done with endotracheal anaesthesia, with opening the trachea when the tube is inside it, the tube should be removed after operation is over.

**Lung cyst** Severity of the patient's condition is due to the collapsed lung, displaced mediastinum, compression of the non-involved lung, and disordered blood circulation

The *preparatory* operations are only few inhalation of oxygen, administration of atropine and cardiac preparations *Anaesthesia* anaesthesia is induced with halothane and oxygen (at high concentration), laryngoscopy without relaxants or after administering depolarizing relaxants, but using no mask for artificial lung ventilation. After intubation of the patient, artificial lung ventilation should be performed without applying high pressure, spontaneous respiration should be restored as soon as possible. This decreases the danger of stomach distension, cyst strain, and, accordingly, compression of the lung and mediastinum. After mobilization of the cyst, artificial lung ventilation can be carried out. If the child's condition is critical, the trachea should be intubated and artificial lung ventilation with minimal inspiratory pressure should be performed. In the absence of adequate lung ventilation *after operation*, controlled respiration should be given to the child at a minimum pressure (15-20 cm H<sub>2</sub>O). Spontaneous respiration with positive expiratory pressure is indicated

**Operations for lung destruction and abscesses, and pleural empyema.** The *preparation* for operation for several hours includes infusion therapy, aspiration of fluids from the pharynx and trachea, and also oxygen therapy. *Anaesthesia* should be endotracheal (twin tubes should be used with older children). If leakage through the fistula is significant, anaesthesia should first be endobronchial, and after the leakage is eliminated the tube may be moved to the trachea. If one-lung anaesthesia is impossible, hypoxia can be lessened by the following techniques: a—by spontaneous respiration, b—by rapid thoracotomy and elimination of leakage, c—by artificial lung ventilation with a short and abrupt inspiration. At the moment of decortication it is desirable to administer procaine into the lung root. Possible blood loss should be replenished. *Post-operative* infusion therapy aimed at detoxication, sanitation of the tracheobronchial tree, and oxygen therapy

**Lung resection** The planned *preparation* of the child for operation includes detoxication and sanitation of the lung and trachea. *Anaesthesia* endotracheal inhalation of nitrous oxide and halothane with oxygen. In older children, especially in lateral thoracotomy,

twin tubes should preferably be used, one-lung anaesthesia should be used for pneumonectomy. Before suturing the pleural cavity, it is necessary to check that the remaining portion of the lung is distended. It is important that the lung should be distended *after operation*. This can be attained by active drainage stimulating the coughing reflex, and spontaneous respiration with positive expiratory pressure.

**Chalasia and achalasia of the oesophagus** *Preparation* of a child to operation is general, because the child is often cachectic. *Anaesthesia* endotracheal with optimum relaxation during the time when manipulations are performed under the diaphragm, in order to prevent reflexes during manipulations on the nervus vagus, it is desirable to ensure procaine infiltration in the region of the nerve, adequate relaxation and ventilation, and the use of  $\beta$ -blockers.

**Oesophagoplasty.** What has just been said of anaesthesia in chalasia and achalasia holds for oesophagoplasty. The *preparatory treatment* includes correction of protein disorders, sanation of the tracheobronchial tree and treatment of chronic pharyngitis. *Anaesthesia* endotracheal with relaxation of muscles. In order to maintain sufficient arterial pressure (prophylaxis of ischaemic disorders in the intestinal transplant), it is reasonable to conduct controlled hypervolaemia, it is therefore necessary to transfuse 50-300 ml of blood and polyglucin before anaesthesia is induced. If the arterial pressure does not elevate by 5-10 mm Hg, hydrocortisone should be administered. The volume of blood transfused during operation should exceed the loss. *Post-operative* prevention of oedema of the subglottic space, effective analgesia, and oxygen therapy.

**Funnel (Cobbler's) chest** There exists a danger of profuse bleeding and of opening of both pleural cavities. The *preparatory measures* are common. *Anaesthesia* endotracheal with muscle relaxants, combined epidural. Replenishment of blood loss. *Post-operative* prolonged intubation and artificial lung ventilation may be indicated for upset respiratory function due to disturbed osteomuscular framework.

**Intestinal obstruction** Water-electrolyte metabolism can be upset due to disordered absorption and vomiting. *Preparatory procedures* due to disordered absorption and vomiting. *Preparatory procedures* infusion therapy (5-10 per cent glucose solution, potassium chloride, sodium bicarbonate, whole blood and plasma, vitamins). Thorough gastric lavage. *Anaesthesia* endotracheal, epidural analgesia. In order to decrease the toxic effect during operation, it is necessary to empty the intestine. *Post-operative* correction of metabolic acidosis, which often intensifies the action of muscle relaxants and retards recovery from anaesthesia.

**Embryonic hernia** Anaesthesia is ordinary if hernia is small. In cases with large hernias, there exists a danger of respiratory and circulatory dysfunction due to abrupt elevation of the intra-abdominal pressure after correction of the hernia. *Anaesthesia* (endotracheal)



should be conducted so that the action of muscle relaxants should discontinue after the hernia is replaced and before suturing. The respiratory and blood circulatory functions can then be tested. Severe respiratory dysfunction and circulatory failure are indications for two-step operations.

**Operations on the stomach and spleen** Endotracheal anaesthesia with nitrous oxide and muscle relaxants, controlled respiration. Muscular relaxation is maintained until the abdominal cavity is sutured. Endotracheal anaesthesia with nitrous oxide in combination with neuroleptanalgesia or epidural anaesthesia should preferably be conducted in weak and asthenic children.

**Hirschsprung's disease** *Preparatory procedures* include correction of upset water-electrolyte and protein metabolism. Premedication includes atropine, promedol and antihistaminic preparations. *Anaesthesia* endotracheal, neuroleptanalgesia, administration of procaine into the mesentery. During manipulations in the small pelvis, anaesthesia should be deepened by additional doses of promedol, phentanyl, and by increasing the concentration of strong anaesthetics. All blood lost should be replenished and measures to prevent shock taken.

In *liver* affections halothane should be rejected and neuroleptanalgesics used instead. *Nephrourological* operations should be carried out under combined endotracheal anaesthesia with fractional administration of muscle relaxants. *Appendectomy, herniotomy*, operations for *cryptorchidism*, and other minor operations on the organs of the abdomen and the small pelvis should be performed with anaesthesia given by the apparatus and mask. Endotracheal anaesthesia is indicated in appendicular infiltration or local *peritonitis*.

**Anal atresia** If operation is short (surgical opening of the anus), anaesthesia is given in a usual way by mask and apparatus. Measures to prevent circulatory disorders during abdominoperineoplasty are the same as in Hirschsprung's disease.

**Operations on the locomotorium** Anaesthesia depends on the extent of surgical intervention, injury, loss of blood, and the position of the child on the operating table. Comparatively small operations can be performed with anaesthesia given to the child lying on his back by mask from the apparatus. Operations associated with significant surgical injury should be performed with multi-component endotracheal anaesthesia. This especially holds for cases where the child lies with face down, on his side, or other nonphysiological positions.

**Anaesthesia in shock** In the presence of bleeding, the surgeon has to perform an operation even if a child is in shock. The operation should be begun with massive infusion therapy (see Chapter 19). For special indications the operation should be preceded by procaine block in the site of fracture, by vagosympathetic block in injuries

of the chest, by paranephric block in injuries of the extremities and the pelvis, etc

Multi-component endotracheal anaesthesia with controlled respiration ensures adequate ventilation of the lungs and good analgesia without inhibiting the respiratory or circulatory centres. Controlled artificial lung ventilation in endotracheal anaesthesia increases efficacy of subsequent measures aimed at restoring normal circulation of blood. Self-regulation of the vital functions is preserved, while sufficient oxygenation of blood has a favourable effect on the condition of the central nervous system, the heart, kidneys, liver, and other organs. This type of anaesthesia in shock can thus be considered a resuscitation measure.

Ketamine and nitrous oxide can be regarded as the most suitable narcotic substances. Neuroleptanalgesia can be used if the blood circulation is stable. Premedication should be minimal and include only necessary substances. Atropine, pipolphen and corticosteroid hormones should be injected intravenously. Morphine analgesics should be rejected for their marked inhibiting effect on the respiratory and vasomotor centres. These preparations can only be used to maintain anaesthesia during operation and during the post-operative period (rather than during premedication of patients in shock). Sodium oxybate, nitrous oxide with oxygen can be used for induction of anaesthesia. It is not recommended to give barbiturates to children in shock, because this preparation can cause disorders in blood circulation.

## Chapter 14

### Hazards and Complications of Anaesthesia in Children

Complications associated with anaesthesia can develop irrespective of adequacy of procedures performed before operation. Every anaesthesiologist should therefore be aware of all side-effects that may occur after anaesthesia, all dangers and complications associated with the use of anaesthetics, possible causes of these complications and measures to prevent and treat them. Correct and timely diagnosis of complications associated with anaesthesia is very important. Permanent control of the child's condition during and after operation facilitates this problem.

Rapidly increasing concentration of halothane in the inhaled gas mixture or prolonged supply of the anaesthetic at high concentration can cause bradycardia (to asystole) and even arrest the heart's action. The danger of these complications is higher in the presence of hypercapnia. The hepatic function can be disordered in the early

post-operative period after a prolonged halothane anaesthesia

A rapidly increasing concentration of ether in the breathing mixture can cause laryngospasm, abrupt cardiac dysfunction, and sometimes asystole due to a strong irritation of the nervus vagus

Cyclopropane can provoke serious disorders in the heart rhythm

Ketamine administered in a dose of 8 mg per kg body weight can cause hyperthermia and a condition during which convulsions are likely to occur in the early post-operative period. Big doses of sodium oxybate can also cause convulsions

Depolarizing muscle relaxants (succinylcholine and its analogues) can cause marked bradycardia (to asystole) in children after the first administration. Repeated administrations of these preparations can cause tachycardia and moderate hypertension

Tubocurarine causes moderate ganglioblocking effect with reduction of arterial pressure

**Respiratory complications.** These can be divided into two groups, namely, respiratory complications in the airways (tracheobronchial tree) and complications in the lungs. All complications associated with obstruction of patency in the tracheobronchial tree provoke a rapidly progressing hypoxia. They can occur during anaesthesia and also during the post-operative period. Mechanical obstruction of the airways with foreign objects, aspiration of the vomited matter, tongue retraction, laryngospasm, bronchospasm, and breakdown in the apparatus are among the most common causes of difficult passage of the gaseous mixture into the alveoli

Various foreign bodies, stomach contents and blood can get into the airways both during induction and maintenance of anaesthesia. The presence of small foreign objects or vomitus can be overlooked during anaesthesia and they can become the cause of serious complications during the post-operative period (strong hypoxia, atelectasis of the lung or its lobe, lung abscess). Signs of asphyxia rapidly develop in severe obstruction of the airways: cyanosis, rapid pulse, elevation of arterial pressure, convulsions are then followed by the arrest of heart's action. The time from the cessation of respiration to arrest of the heart is 3-5 minutes. Decisive measures should be taken during this time to remove the causes of asphyxia

When anaesthesia is administered by mask, it is necessary to be sure that the tongue of the child is not retracted. If obstruction to the air passage is due to tongue retraction, the child's head should be pulled back; this will in most cases remove this complication. If respiration is stertorous, the mandible should be pulled and held in this position. The airway should reach the root of the tongue, otherwise the air delivery will be inefficient. The mouth should then be inspected to see that no vomited matter is present in the mouth and the teeth are intact (they can be broken by clumsy manipulations with a mouth gag or a laryngoscope). The work of the anaesthesia

apparatus should be checked rapidly the oxygen supply to the patient must be adequate, the airway should impose no resistance to respiration, and the inhalation and exhalation valves should function properly

If anaesthesia is administered through an endotracheal tube, the apparatus should be in proper working order, the tube should have no abrupt bends and ensure unobstructed air flow. If the tube is passed too far, the inflated cuff may close a bronchus. Or the tube can pass one bronchus but close the other. Or the aperture of the tube can be closed as the tube end thrusts against the bronchus wall. In order to prevent these complications, it is necessary to auscultate the lungs on both sides, and if respiratory sounds are not heard on one side, the endotracheal tube should be pulled up until the respiratory sounds are well heard over all portions of both the left and right lung.

Accumulation of much mucus or blood in the airways, or penetration of the gastric contents into them, causes hypoxia as well. The tracheobronchial tree should in such cases be immediately cleaned by aspiration, which is simpler in intubation anaesthesia.

When conducting endotracheal anaesthesia, the anaesthesiologist must auscultate periodically the air hoses of the apparatus. Rales indicate the presence of mucus in the bronchi, which should be removed. The tube may be removed from the trachea only after complete restoration of spontaneous respiration and the coughing reflex. Before removing the tube, the contents of the bronchi and trachea should be removed thoroughly by aspiration, and the mouth then dried up.

Laryngo- or bronchospasm is possible if mechanical obstacles are absent while controlled respiration is difficult because of the high resistance to inspiration. These complications are infrequent provided anaesthesia is given properly. *Laryngospasm* usually develops through reflex routes in response to irritation of the mucosa of the upper airways with concentrated vapours of anaesthetics, coarse manipulations with a laryngoscope, especially if induction anaesthesia is insufficiently deep or the dose of a relaxant is small. It may also occur in response to an attempt to introduce the airway before the laryngeal reflex is depressed, during operations on the abdominal cavity with general or local anaesthesia (when the stomach is pulled), during irritation of the solar plexus, and other rough manipulations associated with irritation of the branches of the *nervus vagus*. When barbiturates are infused rapidly, laryngospasm can occur due to increased tone of the *nervus vagus*. Removal of the tube can also cause laryngospasm, if performed clumsily.

If the vocal slit is closed incompletely, a specific whistling sound can be heard during inhalation. While respiration is spontaneous, the tidal volume decreases markedly. This becomes obvious by the decreased amplitude of the air bag movements despite energetic re-

spiratory excursions of the chest. A complete laryngospasm arrests the air supply to the lungs and even increased pressure on the air bag does not help. Laryngospasm often abates spontaneously and quickly. But in some cases it can last for a long time, while the signs of hypoxia increase rapidly. The cause of laryngospasm should in the first instance be removed as soon as possible. If laryngospasm occurs in the early stage of anaesthesia, the anaesthetic supply should be discontinued and the patient should be given pure oxygen to breathe. If this measure fails, muscle relaxants should be administered with subsequent intubation. If laryngospasm develops during operation, the surgical intervention should be suspended and atropine solution administered into the vein, anaesthesia should be deepened. Tracheotomy should be performed in extreme cases, if laryngospasm persists and attempts to conduct intubation fail.

*Bronchospasm* is a more severe complication, which interferes with delivery of the breathing gas into the lungs. Its causes are the same as of laryngospasm. Bronchospasm occurs mostly in children with chronic lung diseases and allergic reactions. But in some cases this complication occurs also in patients without pulmonary pathology. Bronchospasm is usually provoked by parasympathomimetic substances (barbiturates, cyclopropane), especially in the absence of adequate premedication. Severe contraction of the bronchi (to their complete obstruction) can occur at any period of anaesthesia, but it usually occurs during induction.

Bronchospasm is attended by severe hypoxia, the respiratory excursions of the chest are discontinued, the patient's face turns greyish, the lips are cyanotic, the pulse accelerates and weakens. If bronchospasm occurs in an intubated child, controlled respiration becomes difficult and air is expressed from the bag only by a great effort, while exhalation becomes long, coarse dry rales can be heard. Laryngo- and bronchospasm sometimes occur simultaneously.

Injected intravenously are atropine, diaphylline or euphylline, droperidol, hydrocortisone, calcium chloride, a 4 per cent sodium bicarbonate solution, vagosympathetic block is carried out. Anaesthesia should be deepened whenever possible. These measures can prove ineffective in total bronchospasm. Smolnikov's open chest massage of the lung is used as an extreme measure in such cases. A draining tube is passed into the pleural cavity and connected to another anaesthesia apparatus. The anaesthesiologist presses alternately the bags of both apparatuses to pump air into the pleural cavity or the lungs (at a rate of 10-12 per minute). All medical manipulations must be sufficiently rapid because the heart can stop at any moment of developing hypoxia.

If treatment of bronchospasm is ineffective and air is absorbed in the alveoli, much fluid is liberated from the capillaries and the lungs are affected by *oedema*. Oedema can also be caused by an abrupt ele-

vation of arterial pressure during anaesthesia, hypertension in the lesser circulation, infusion of excessive liquid, or incorrectly controlled respiration. Oedema of the lung can also occur in the early post-operative period. This is an extremely severe complication of anaesthesia, which is very difficult to manage. Clinically it is manifested by increasing cyanosis, noisy and gurgling breathing, moist rales, and pinky foaming in the mouth.

The operation must be suspended in the presence of signs of lung oedema. Intensive therapy for lung oedema is described in Chapter 18.

Mechanical obstruction of the bronchus or its significant contraction caused by the reflex effect of the surgical injury on the vagosympathetic system upset the bronchial conduction and a part of the lung is not involved in the respiratory function. Air is soon absorbed and a part of the pulmonary tissue, not involved in the respiratory act, collapses (*atelectasis*). This complication is markedly more frequent than actually diagnosed. Reflex atelectasis can occur during the operation as well. The diagnosis then becomes especially difficult because only small portions of the lungs are affected and hypoxia is absent because of the high oxygen concentration in the anaesthetic mixture. If respiratory distress is obvious, it can erroneously be explained by some other cause, e.g. acute cardiovascular failure, overdosage of narcotics, shock, etc.

Treatment of patients with atelectasis is described in Chapter 17.

**Cardiovascular complications.** A sudden *arrest of the heart's action* is the most serious complication that can occur in any stage of anaesthesia and in early post-operative period. The term 'sudden' is not quite correct, because a skilled and careful anaesthesiologist will always identify the precursors of the heart's arrest by the changes in the patient's condition (blood circulation included). Early diagnosis of these symptoms and their timely correction promote the restoration of normal cardiovascular function and prevent a 'sudden' cessation of the heart's action. Besides, inadequate cardiovascular function before operation is a direct indication that various circulatory disorders are likely to happen during anaesthesia. In such cases the control of the patient's condition must be especially careful. Arterial pressure and pulse should be taken at short intervals, the colour of the skin and blood in the operative wound should be observed attentively. The condition of the patient should be monitored.

The precursors of acute cardiovascular failure are progressive tachy- or bradycardia, cardiac arrhythmias, irregular respiration, a sudden pallor or cyanosis of the skin, slowly developing or a sudden hypotension, and dark blood. Electrocardiographic control of the patient's cardiovascular function during operation is a valuable means. It is especially important in patients with concurrent diseases of the cardiovascular system.

The heart can stop because of hypoxia of various aetiology, acute blood loss during operation, narcotic overdosage, rapid administration of barbiturates (especially to asthenic patients), acute adrenal insufficiency, excessive irritation of reflexogenic zones and organs richly innervated with endings of the nervus vagus. The heart can stop by reflex during intubation of the trachea.

If signs of circulatory insufficiency (hypotension, cyanosis, weak pulse, etc.) develop during operation, measures should immediately be taken to improve the myocardial contractility (strophanthin with glucose and vitamins, cocarboxylase, ATP). Since the heart often stops during hypoxia and hypercapnia, adequate lung ventilation must be ensured.

If pulse is absent and arterial pressure falls to zero, the diagnosis of cardiac arrest is established, although very weak contractions of the heart can be detected in some cases by the monitor. Energetic therapy should then be undertaken urgently. Anaesthetic supply should be discontinued and artificial respiration with pure oxygen given.

Resuscitation of patients with stopped heart are described in Chapter 18.

**Heart rate and rhythm disorders.** During early anaesthesia pulse can be accelerated due to a psychic excitation of a child before he is taken to the operating room. The heart rate normalizes in such cases spontaneously (without any treatment). The heart rate can also increase during induction anaesthesia, but as the narcotic sleep deepens, the heart rate normalizes. Transient tachycardia with elevated arterial pressure usually occurs immediately after intubation in response to a short period of hypoxia (during intubation). As soon as the patient's lungs are ventilated artificially, the heart rate and arterial pressure normalize. In all other cases accelerated pulse is the sign of severe condition and indicates the necessity of immediate treatment.

The most frequent causes of tachycardia during operation and early post-operative period are the onset of hypoxia, bleeding, shock, injurious surgery, early overdosage of anaesthetics. Excessively shallow anaesthesia also causes tachycardia. If pulse is accelerated by acute blood loss or developing shock, it is necessary to transfuse blood, polyglucin or plasma. If signs of hypoxia develop, measures to improve lung ventilation should be taken. Pronounced tachycardia occurring after anaesthesia (if it is not attended by an abrupt elevation of body temperature) can also be the result of respiratory distress, post-operative shock, non-compensated blood loss, or reaction to severe pain in the surgical wound. Measures to correct this condition depend on the cause.

Heart rate decreases if the tone of nervus vagus increases due to hypoxia or the nerve is irritated during operations on the lungs and

the upper abdomen. Marked bradycardia can occur due to intravenous administration of proserine, which is used as an antidote to non-depolarizing muscle relaxants. Strong bradycardia is treated by intravenous administration of atropine. But if bradycardia develops in the presence of hypoxia or due to overdosage of narcotics, the use of this particular preparation should be suspended and measures taken to eliminate the disorders produced.

Various types of arrhythmia occur during and after anaesthesia. As a rule, arrhythmia can be diagnosed by common methods of examination. Arrhythmia can be caused by myocardial hypoxia, hypercapnia, by changes in the myocardium caused by concurrent diseases, toxic effects of some anaesthetics, excessively deep anaesthesia, hyperkalemia, or acidosis.

**Hypo- and hypertonic reactions.** Fall of the arterial pressure during operation and anaesthesia or during early post-operative period is a common complication. It can be caused by hypoxia, poisoning, overdosage of narcotics, acute blood loss, shock, heart failure due to myocarditis or other diseases, or acute adrenal failure. If hypotension is not attended by acceleration or deceleration of pulse rate, it is due to decreased tone of the peripheral vessels and is usually abated by vasopressor preparations. Transfusion of blood or its substitutes is less effective in such cases. But the arterial pressure normalizes for a short period and hypotension may develop again, especially in children with chronic diseases or in children treated with steroid hormones. Adrenocortical insufficiency can be suggested in such cases. Intravenous administration of polyglucin with nor-epinephrine and hydrocortisone is a reliable means against this complication.

If arterial pressure falls as a result of shock, blood loss, poisoning, deep anaesthesia, or overdosage, the heart rate accelerates markedly. Treatment should then be aetiological. Hypotension with bradycardia occurs much less frequently. It is caused by excessive depression of the vasomotor centre by narcotic substances or increased tone of the nervus vagus in the presence of hypoxia, which impairs the contractility of the myocardium. Hypotension of this type occurs also in deep halothane anaesthesia. Substances increasing excitability of the respiratory and vasomotor centres, atropine, glucose with vitamins, and oxygen inhalation are used to manage this condition.

Arterial pressure increases if anaesthesia is inadequate. This occurs frequently in nitrous oxide anaesthesia, early hypoxia and hypercapnia. If hypertension is due to oxygen deficiency or hypercapnia, lung ventilation should be improved. Shallow anaesthesia should be deepened. If nitrous oxide is used as an anaesthetic, halothane may be added during operation's most dramatic moments.

**Malignant hyperpyrexia** is a rare but very dangerous complication. Its aetiology is unknown. Depolarizing muscle relaxants



and, possibly, genetically-determined factors may be involved. This complication manifests clinically by an abrupt elevation of body temperature to 39-40°C, hyperaemia of the skin, and upset respiratory and circulatory function (tachycardia, hypotension). Malignant hyperpyrexia can rapidly cause oedema of the brain and death. Treatment of this syndrome is described in Chapter 23.

## Chapter 15

### Anaesthesia of Premature Neonates and of Neonates Born at Term

Anaesthesia of neonates, and especially of those delivered prematurely, is a complicated aspect of anaesthesiology and intensive therapy. During the first 10 or 12 days of life the state of a neonate can be characterized as a post-natal stress. It is quite natural therefore that diseases requiring surgical correction increase the risk factor.

A neonate should be anaesthetized by a specially trained physician, who must be a skilled anaesthesiologist and resuscitator and must know very well the fundamentals of neonatology.

When a neonate is transported into or from the operating room, measures must be taken to prevent his cooling. A special operating room must be provided for neonates, where the ambient temperature should be maintained not below 25°C, and the operating table must be provided with a special heater. The neonate should be brought into the operating room in a couveuse when everything is prepared for anaesthesia and operation. The apparatus for anaesthesia must be provided with all accessories that may be required during operation.

The type of anaesthesia depends on many conditions: the state of the neonate, the character of operation, and experience of the anaesthesiologist. Various types of anaesthesia can be used with neonates but it is desirable that minimum medicinal preparations should be used if possible.

If inhalation anaesthesia is used, endotracheal route should be preferred because a neonate has a relatively large tongue and giving anaesthesia by a mask may be difficult. Special measures should be taken to minimize the resistance of the anaesthesia apparatus, connecting tubings and the endotracheal tube.

A valveless system is used for anaesthesia of neonates, in which the dead space is practically absent and the resistance to respiration is minimal.

The disadvantage of valveless systems is that a child breathes in dry and cold gas. The gas mixture should therefore be humidified and warmed for artificial ventilation of the lungs.

Curved endotracheal tubes without cuffs are used for intubation.

of neonates. It is convenient to use endotracheal tubes in which the proximal end has a larger diameter (to prevent its deep passage into the trachea).

The use of connectors should be avoided since they increase the dead space and resistance of the system.

As distinct from older children, neonates need no anaesthesia or muscle relaxants for *intubation of the trachea*. Weak neonates (with hypotrophy, cachexia, muscular hypotonia, hyporeflexia) or premature neonates can be intubated before giving anaesthesia (after premedication). Neonates with adequate muscular tone and normal reflexes are intubated under anaesthesia after administering depolarizing muscle relaxants. Patients whose condition is intermediate between these two groups should be intubated under anaesthesia after a short hyperventilation.

*Anaesthesia* of neonates is usually *induced* by inhalation anaesthetics because their effect is easier to control. But in some cases where multi-component anaesthesia with neuroleptanalgesics or sodium oxybate is indicated, anaesthesia is induced intravenously (intramuscularly with ketamine).

Depending on indications and technical facilities, several variants of induction anaesthesia are possible in neonates, e.g., nitrous oxide with oxygen + halothane, tracheal intubation + nitrous oxide with oxygen + halothane, or promedol, nitrous oxide with oxygen + halothane, or cyclopropane + tracheal intubation, nitrous oxide with oxygen + halothane + muscle relaxants + tracheal intubation, neuroleptanalgesics + nitrous oxide with oxygen + muscle relaxants + tracheal intubation, ketamine + nitrous oxide with oxygen + muscle relaxants + tracheal intubation.

It is quite natural that other variants are possible depending on experience and skill of the anaesthesiologist, presence or absence of particular medicinal preparations, and special indications.

*Maintenance of anaesthesia* begins after the end of the induction period and terminates with signs of recovery. Type of anaesthesia for neonates, as well as for all other patients, should be selected depending on the initial condition of the patient, the character and length of operation, and skill of the anaesthesiologist. It is necessary to remember that objective and accurate assessment of the neonate's condition is much more difficult than of an adult, and therefore, when selecting the type of anaesthesia, it is necessary to minimize the effect of various factors on the child.

Multi-component general anaesthesia is commonly used with neonates. Simple one-component anaesthesia is less frequent, and still less frequently used is local (infiltration) anaesthesia.

**One-component anaesthesia with nitrous oxide.** Anaesthesia is induced and maintained with nitrous oxide and oxygen delivered in the ratio of 2:1 or 3:1 (endotracheally or by mask).

Indications small extracavitary operations (incisions, opening of abscesses, puncturing of cavities or vessels, endoscopy, painful dressing)

**One-component anaesthesia with halothane.** Anaesthesia is induced and maintained with halothane and oxygen. The concentration of the anaesthetic is increased gradually to the level not exceeding 0.015 l/l (1.5 per cent v/v). Anaesthesia is given endotracheally or by the mask. The indications are the same as for nitrous oxide anaesthesia. Contraindications physiological and other jaundice, and disorders in myocardial contractility.

**Multi-component anaesthesia with nitrous oxide and halothane.** Anaesthesia is induced and maintained with nitrous oxide, oxygen and halothane. Anaesthesia can be given with or without muscular relaxation, endotracheally or by the mask. Indications most operations. Contraindications diseases of the liver and severe circulatory disorders.

**Multi-component anaesthesia with neuroleptanalgesics.** Induction anaesthesia droperidol (0.3 mg/kg) and phentanyl (0.008-0.01 mg/kg) intravenously, nitrous oxide and oxygen (2:1) and muscle relaxants (tracheal intubation). Anaesthesia is maintained with nitrous oxide and oxygen (1:1) and phentanyl in decreasing ( $1/2$ - $1/3$  of initial) doses. Indications prolonged injurious operations in children with hepatic dysfunction and complications on the part of the cardiovascular system. Contraindications markedly decreased volume of circulating blood and pronounced dehydration.

**Multi-component anaesthesia with ketamine.** Induction anaesthesia ketamine intramuscularly in a dose of 10-12 mg/kg. Anaesthesia is maintained with nitrous oxide and oxygen (2:1), endotracheally or by the mask. Indications injurious operations, decreased myocardial contractility, hypovolaemia, low arterial pressure. Contraindications hyperhydration and hypertension. Ketamine can be used for multi-component anaesthesia with nitrous oxide and halothane. Ketamine is administered intramuscularly in a dose of 6-7 mg/kg; the concentration of halothane should not exceed 0.01-0.015 l/l (1-1.5 per cent v/v).

**Multi-component epidural anaesthesia.** The epidural space of neonates is punctured and catheterized as in children of other age. A 1-1.5 per cent trimecaine solution is used for epidural anaesthesia of neonates in a dose of 15-20 mg/kg. In any case the dose should not exceed 4 ml per single injection. A fall in the arterial pressure during epidural anaesthesia, which is reported by most authors, is observed in neonates as well. But if the initial blood volume is normal and the rate of trimecaine administration is 1 ml within 2 minutes, the fall in the arterial pressure does not usually exceed 5-8 per cent of the initial, the pressure is restored to the pre-operative level in 20 minutes after the administration. The arterial pressure thus returns to

the initial level by the moment the operation is started. The anaesthetic should be administered repeatedly in 1 hour ( $2/3-1/2$  of initial dose).

Epidural anaesthesia can be used independently during correction of congenital growth defects of the gastrointestinal tract. Neonates behave quietly during operation and often fall asleep. Adequate relaxation of the abdominal wall provides good conditions for manipulations in the abdominal cavity. Multi-component epidural anaesthesia is indicated for teratoma of the sacrococcygeal region, intestinal obstruction, peritonitis, and diaphragmatic hernia in neonates.

The neonate's condition is controlled and his homeostasis during operation is maintained as in children of other age. But as distinct from older children, neonates should be auscultated (along with monitoring) to follow the character of heart sounds and the pulse on the peripheral arteries. The anaesthesiologist can thus detect the early signs of disorders in the heart's action and prevent their progress. An oesophageal phonendoscope is suitable for the purpose.

The loss of liquid during operation should obligatorily be compensated completely in neonates. It has been found that in order to correct the liquid deficit, which is not associated with the blood loss (if the surgical injury is not vast), not less than 4-6 ml/kg per hour of rheopolyglucin or a 10 per cent glucose solution with Ringer's solution (1:1) should be administered. If the injury is vast, the rate of administration should be not lower than 8-10 ml/kg per hour. The blood loss should be restored completely but not obligatorily with whole blood; in some cases blood substitutes should be preferred. If the initial haemoglobin content was high and the blood loss during the operation does not exceed 10 per cent of the circulating volume, blood transfusion is not obligatory. Blood extenders, glucose and salt solutions are used instead. If the blood loss is from 10 to 20 per cent of the circulating volume, 50 per cent of the loss is replenished by whole blood and 50 per cent by other solutions. If the blood loss exceeds 20 per cent and more,  $2/3$  should be whole blood and  $1/3$  blood extenders and solutions. It should be remembered that freshly-citrated blood (stored for not more than three days) may only be transfused to neonates (especially during their first week of life).

Body temperature is very important. It is necessary to remember that subnormal temperature which is common in neonates during anaesthesia and operation can become the cause of many complications in the early post-operative period. The breathing gas given for anaesthesia should therefore not only be humidified but also warmed up; the temperature of solutions for transfusions should be  $37^{\circ}\text{C}$ .

*Recovery from anaesthesia* is a very important stage. Spontaneous respiration is restored in neonates slower than in older children. But

if anaesthesia was adequate (anaesthetics or muscle relaxants were not overdosed), spontaneous respiration is usually restored soon after discontinuation of artificial lung ventilation. If respiration remains shallow and irregular, it is necessary to continue artificial ventilation of the lungs until spontaneous respiration is adequate. If the operation was long and injurious and if promedol and neuroleptanalgesics were used as anaesthetics, artificial lung ventilation should be continued in the intensive therapy unit, the transition to spontaneous respiration is gradual. When the child begins breathing spontaneously, the Gregory method (constant positive airway pressure) should be used with constant control of the blood gas tension and with observation of the child by the physician. As soon as spontaneous respiration is restored, it is very important to see that the airways are free, which is important to prevent respiratory distress (hypoventilation, atelectasis, pneumonia, and the like).

Infusion therapy, which begins immediately after operation, is a logical continuation of the therapy that was begun during the pre-operative period and during anaesthesia. The therapy is, first of all, directed at correcting circulatory disorders and also water-electrolyte and acid-base balance.

A special couveuse is used to transport a neonate into the hospital and from the operating room. Special temperature and humidity conditions are maintained in the couveuse and its atmosphere is enriched with oxygen.

Severe disorders in the vital functions are likely to occur in an infant during recovery from anaesthesia after vast and injurious operations. The danger even more increases since all disorders form a kind of a vicious circle, where dysfunction of one system involves disorders in haemostasis. All methods of intensive therapy should therefore be used during recovery from anaesthesia and they all should be aimed at correcting respiration, blood circulation, metabolism, energy and temperature balance, and other body functions. The main task of the anaesthesiologist is to prevent possible disorders.

### SOME SPECIAL CASES OF ANAESTHESIA

**Atresia of oesophagus** The amount of preparatory measures depends on the gravity of the neonate's condition. If a defect is revealed during the first hours of the neonate's life (in the absence of concurrent defects) and the respiratory function is normal, the preparatory measure includes only placing the neonate in a couveuse (elevated head end) where special climatic conditions are maintained. The proximal end of the oesophagus and the oropharynx should be constantly cleaned by aspiration. The operation should be performed practically immediately as soon as the blood group and the Rhesus factor have been determined. No premedication is required.

If oesophageal atresia (especially with tracheo-oesophageal fistula) is not diagnosed in time, the neonate usually develops marked aspiration pneumonia, respiratory distress, hypovolaemia, and dehydration. The pre-operative procedures usually take 24-72 hours. The neonate is placed in a couveuse with an elevated head end. The oropharynx, the proximal end of the oesophagus and the tracheobronchial tree are constantly cleaned. The neonate is turned from side to side at 30-minute intervals. Inhalations and percussion massage are done at 60-minute or shorter intervals. If hypoxaemia due to aspiration pneumonia is significant, it is recommended to conduct oxygen therapy under positive pressure. The Gregory method should be preferred because the danger of penetration of the gastric contents into the bronchial tree is thus decreased. The neonate should simultaneously be examined and given infusion therapy to correct volaemic and microcirculatory disorders, to eliminate dehydration and to normalize acid-base balance and metabolism. This is attained by glucose, albumin, blood plasma, rheopolyglucin, and potassium chloride. Broad-spectrum antibiotics, preparations improving metabolic processes in the myocardium and immune processes in the body are administered 40 minutes before anaesthesia, 0.2-0.3 ml of vikasol is injected intramuscularly.

Anaesthesia is induced either using the apparatus with the mask by which halothane (0.01 l/l or 1 per cent v/v) with nitrous oxide and oxygen (2 l) are administered, or by intravenous administration of sodium oxybate (100 mg/kg) and promedol (0.3 mg/kg). The trachea is intubated after administering of 2 mg/kg depolarizing muscle relaxants (premature and asthenic neonates can be intubated without relaxation of their muscles).

The lungs are ventilated by a semi-open method (the Ayre-Rees modification). Anaesthesia is maintained by breathing halothane (0.004-0.005 l/l or 0.4-0.5 per cent v/v) with nitrous oxide and oxygen (1 l or 2 l), or by neuroleptanalgesics.

Liquid loss is replenished at a rate of 8-10 ml/kg per hour, blood loss should be replenished completely.

After operation the neonate is transported to the ward where he breathes oxygen through the tube, which is not removed after operation, under constant positive airway pressure (Gregory method). All measures taken during the early post-operative period are a logical continuation of measures that were taken during the pre-operative period, during the operation and anaesthesia. During the first two days following the operation, the neonate is given total parenteral nutrition: 2 g/kg protein, 4 g/kg fat and 12-14 g/kg carbohydrates. On the second day (in the absence of congestion in the stomach) the neonate should be given 5 ml of a 5 per cent glucose solution at 2-hour intervals (through the tube). On the third day the neonate is given 5 ml of breast milk at two-hour intervals, this portion should

be increased daily. As the amount of food given per os increases, the amount of nutrients given parenterally should be decreased accordingly.

**Congenital diaphragmatic hernia** The type of anaesthesia during operations for diaphragmatic hernia in neonates depends on disorders in gas exchange and blood circulation caused by the collapse of the involved lung and impaired gas exchange in the intact lung as a result of displacement of the mediastinal organs to the opposite side, this is manifested by acute respiratory and cardiovascular failure. The earlier the signs of these disorders develop, the worse the prognosis. Since the abdominal organs are displaced into the thoracic cavity, the ventilation of the lungs cannot be improved and the circulatory disorders cannot be corrected. Pre-operative measures are thus useless, moreover, they may be dangerous.

Premedication, induction and maintenance of anaesthesia are practically the same as during operations for oesophageal atresia. In all cases the trachea is intubated after administration of depolarizing muscle relaxants. It should be remembered that after the administration of muscle relaxants, hyperventilation of the lungs should be carried out very carefully and with a small tidal volume because forced respiration can cause a rupture of the hypoplastic lung and pneumothorax. This should also be borne in mind when performing artificial ventilation of the lungs to maintain anaesthesia, adequate minute ventilation should be ensured by increasing the respiration rate rather than the tidal volume. The specific feature of anaesthesia during this operation is that effective relaxation of muscles is necessary to ensure better and less traumatic conditions for replacement of the stomach, intestine, spleen, and the liver into the abdominal cavity.

Spontaneous respiration is not always adequate during early post-operative period and prolonged artificial ventilation of the lungs is therefore required. The maximum inhalation pressure should not exceed 20 cm H<sub>2</sub>O because of the danger of rupture of the hypoplastic lung.

When spontaneous respiration is restored it is recommended to maintain constant positive pressure in the airways for several hours. The resistance to expiration should not exceed 5 cm H<sub>2</sub>O. It is important to control possible restrictive changes in the respiration and gas exchange that can be due to increased intra-abdominal pressure caused by displacement of some organs from the pleural cavity into the underdeveloped abdominal cavity. It is especially important therefore to prevent intestinal paresis in such patients. This can be attained with epidural anaesthesia, potassium preparations, proserine, and hypertonic enemas. Enteral nutrition should be completely suspended for at least 3 or 5 days, all energy expenditures should be replenished parenterally.

Preparations controlling hypertension in the lesser circulation should be administered during the post-operative period.

**Lobar emphysema.** The development of the valvular mechanism in the bronchi of the involved lobe, the absence of cartilage in them, and also underdevelopment of elastic tissue in the alveolar walls cause the rapid distension of the involved lung lobe. It occupies a large volume and displaces healthy portions of the lung to compress the non-involved lung portions, thus impairing their ventilation. Hypercapnia and hypoxaemia rapidly develop. Displacement of the mediastinal organs to the opposite side and compression of the large vessels intensify hypoxia and provoke cardiovascular failure.

In emergency cases operations should be performed immediately after the diagnosis has been established. No premedication is therefore carried out. Anaesthesia should be done by the same method (as described above). The special feature is that the trachea is intubated only when the surgeon is ready to perform thoracotomy. This is explained by the danger of overdistension of the emphysematous lobe during artificial ventilation of the lungs with subsequent abrupt impairment of the heart's action. Symptomatic therapy is indicated during the earliest post-operative period. Nutrition can be given per os only in 6 hours after termination of anaesthesia.

**Pylorostenosis.** The preparatory measures include infusion therapy, which is mainly aimed at removal of dehydration, circulatory disorders, correction of the acid-base balance, and the protein and energy metabolism. Duration of pre-operative treatment depends on the gravity of the patient's condition and varies between 24 and 72 hours. The gastric contents should constantly be removed by aspiration with subsequent compensation of liquid and electrolyte loss by infusion of salt solutions. Symptomatic treatment should be carried out for indications. This should be aimed at eliminating the aspiration syndrome.

Following premedication, immediately before giving anaesthesia, the stomach should be lavaged, the tube should not be removed from the stomach after lavaging. The type of anaesthesia depends on the skill of the anaesthesiologist. In the absence of a skilled anaesthesiologist, an operation can be performed with local infiltration anaesthesia using a 0.25 per cent procaine solution. In order to quiet an infant, he is given a soother wetted in a glucose solution with alcohol. Pylorotomy can be done with epidural anaesthesia, one-component ketamine anaesthesia (10 mg/kg intramuscularly), halothane in the concentration of 0.01-0.015 l/l (1-1.5 per cent v/v) with nitrous oxide and oxygen (2:1). This gaseous mixture ensures adequate relaxation of muscles without interfering with the respiratory function, anaesthesia can thus be given by face mask.

The therapy during the earliest post-operative period is a continuation of the therapy that was begun before operation aimed at cor-



recting the water-electrolyte, protein and energy metabolism, and acid-base balance

The infant should be placed with his head in an elevated position in an oxygen tent. Nutrition begins on the next day 5 ml of breast milk at 2-hour intervals, the portion daily increased not more than by 10 ml. The protein and caloric demands are fulfilled by parenteral nutrition.

**Congenital complete intestinal obstruction.** The amount of pre-operative treatment depends on the degree of circulatory disorders, disorders in water-electrolyte metabolism, the degree of dehydration and clinical manifestations of circulatory disorders in the intestinal wall. In the presence of signs of intestinal strangulation with upset circulation, the pre-operative treatment should not last more than 4 hours. If these symptoms are absent, the pre-operative procedures may last for 24-36 hours. The neonate should immediately be placed in a couveuse (after preliminary intubation of the stomach and its irrigation). In the presence of clinical signs of aspiration pneumonia, oxygen therapy should be conducted in a plastic bag with constant positive pressure.

Pre-operative infusion therapy should in the first instance be directed at normalization of macro- and microcirculation (solutions of albumin, plasma, poly- and rheopolyglucin). Next, a glucose solution containing concentrated solutions of electrolytes for correction of the water-electrolyte balance should be infused. The amount and composition of these solutions depend on the degree and type of dehydration and the time allotted for preparation for anaesthesia and operation.

Premedication is common (40 minutes before anaesthesia). Before anaesthetizing the neonate, it is necessary to make sure that his stomach is empty. Anaesthesia should be conducted with endotracheal intubation (Ayre system). Induction anaesthesia should be done with the aid of apparatus and mask (halothane, nitrous oxide, oxygen) or intravenously (sodium oxybate, diazepam, promedol). The trachea should be intubated after administration of depolarizing muscle relaxants (except premature and weak neonates).

Anaesthesia is maintained by various methods (epidural anaesthesia, neuroleptanalgesics, halothane + nitrous oxide + promedol). Any type of anaesthesia, except epidural, should be supplemented with a 0.25 per cent procaine solution injected into the mesenteric root. The concentration of the main anaesthetic (or the dose of the repeatedly administered analgesic) can thus be decreased and relaxation of muscles improved.

During operation liquids should be infused at a rate of 10-12 ml/kg per hour because much liquid is evaporated from the surfaces of the open abdominal cavity (greater than during any other operations). Blood loss should be replenished.

After operation the neonate should be kept in the special climatic conditions again. If the operation was small and not injurious (correction of torsion, incision of commissures), the neonate can be given breast milk (5 ml) immediately after peristalsis of the intestine is restored (usually on the second day after operation). Infusion therapy should comply with the type and degree of dehydration.

Management of patients after resection of part of the intestine is much more difficult. Decompression and drainage of the gastrointestinal tract above the point of anastomosis are important measures. The amount and composition of the fluids lost should be accurately determined and replenished. Measures against possible paresis of the gastrointestinal tract are obligatory: epidural anaesthesia, potassium preparations, proserine, hypertonic enema, and hyperbaric oxygenation. Peristalsis restores and anastomosis begins functioning usually in 3-5 days (sometimes later). During this period of time nutrition should be parenteral: protein hydrolysates or amino acid mixtures, fat emulsions, carbohydrates. The energy loss should be replenished by consuming 120-140 kcal/kg a day. Giving food per os can only be started after development of satisfactory peristalsis and after congestive material is no longer discharged from the stomach.

Infusion therapy should be started from the very first post-operative hours. It is a logical continuation of the therapy that took place during the pre-operative period and during operation.

**Peritonitis.** Pre-operative treatment of neonates with peritonitis usually lasts for 2-4 hours and is directed at control of intoxication, correction of haemodynamic, water-electrolyte and metabolic disorders. The object of the pre-operative treatment is not to eliminate the disorders (only surgical operation can do it), but rather to improve homeostasis in order to minimize the risk associated with anaesthesia and operation.

The stomach of the neonate should be emptied through a gastric tube, which should remain in the stomach till the end of operation. Liquids should be infused at a rate of 10-15 ml/kg per hour. The infused liquids are glucose, hemodes or neocompensan, rheopolyglucin, albumin, or plasma. Moreover, symptomatic therapy to improve the action of the heart and the sympatho-adrenal function should be carried out. Analgesics and antibacterial preparations should also be administered.

Anaesthesia in operations for peritonitis does not practically differ from that for intestinal obstruction. Endotracheal multi-component anaesthesia should be preferred (nitrous oxide with oxygen in the ratio of 1:1, in combination with epidural anaesthesia). Immediately after termination of operation and recovery from anaesthesia, artificial lung ventilation for 3-6 hours is sometimes necessary. When the patient needs artificial ventilation no longer, his spontaneous res-

piration should continue for 2-4 hours under constant positive pressure in the airways

Infusion therapy is a logical continuation of the therapy that was begun before the surgical operation. It is used to control intoxication, volaemic and microcirculatory disorders, water-electrolyte and metabolic disorders in the body. If the renal function is normal, the treatment should include forced diuresis with hourly control of urinary discharge.

Intestinal paresis and parenteral nutrition are controlled as in neonates with congenital intestinal obstruction.

Glycosides, preparations improving metabolism in the heart muscle (cocarboxylase, panangin, calcium chloride, unithiol, complamin) are used for cardiac disorders. Direct blood transfusions are used to stimulate the protective forces of the body. Enteral nutrition begins with 5 ml of breast milk (only after restoration of peristalsis and free patency of the gastrointestinal tract).

**Umbilical hernia** No pre-operative treatment is required because neonates should be operated during the first hours of their life. The neonate should be kept in a couveuse. An elastic tube should be passed into the stomach before operation. Symptomatic therapy can be given if necessary. Premedication and induction anaesthesia are common. The trachea is usually intubated without muscle relaxants. The lungs should be ventilated artificially according to the Ayre method. Anaesthesia should be maintained with nitrous oxide, oxygen and halothane, or epidural anaesthesia. It should be remembered that maximum relaxation of muscles is required when repositioning the protruding parts into the abdominal cavity. Muscle relaxants are therefore often used.

The early post-operative period is characterized by adaptation of the neonate to the high intra-abdominal pressure and hence to restrictive respiratory disorders. During the first few days the neonate is fed parenterally, epidural anaesthesia and sessions of hyperbaric oxygenation are also carried out to promote a rapid resolution of intestinal paresis. Food is given per os only after restoration of the intestinal peristalsis. Nutrition includes 5 ml of breast milk at 2-hour intervals with increasing the daily dose not more than by 10 ml.

## PART THREE

# Resuscitation and Intensive Therapy

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### Chapter 16 Resuscitation

Resuscitation is revival or restoration of vital functions of patients in terminal states or clinical death

#### TERMINAL STATES AND CLINICAL DEATH

Terminal state is the final period of fading life, which precedes biological death. The critical level of vital dysfunctions with a fatal impairment of the heart's action, profound disorders in gas exchange and metabolism is usually called the terminal state. Cardiac arrest with complete cessation of respiration is called clinical death. Biological death is characterized by irreversible changes in the bodily organs and tissues, and, in the first instance, in the central nervous system. Depending on the preceding condition of an infant and the causes of death, terminal states may last for various periods of time and include the following stages: preagonal state, agony and clinical death (Fig 30).

The *preagonal state* is characterized by the general inhibition, fall of the arterial pressure below 60 mm Hg, accelerated and small pulse, dyspnoea, changes in the skin colour (pallor, cyanosis). The preagonal condition lasts from a few minutes to several hours (to one day). This period is characterized by circulatory and respiratory disorders, hypoxia of organs and tissues, and accumulation of underoxidized products. This in turn causes marked disorders in all organs and systems of the body. Agony develops.

*Agony* is the condition characterized by the absence of consciousness and eye reflexes. The heart sounds are dull. Arterial pressure is undeterminable. The pulse of the peripheral vessels is thready and impalpable, on the carotids, it is weak. Respiration is rare and convulsive, or deep and fast. The agonal state lasts from a few minutes to several hours. During agony the last compensatory and adaptive functions of man are often activated, the fading respiration and blood circulation are often reactivated and consciousness can be regained for a short period of time. A rapid accumulation of underoxidized

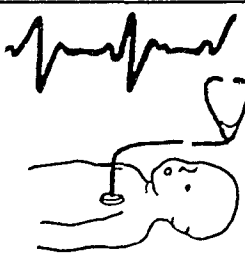
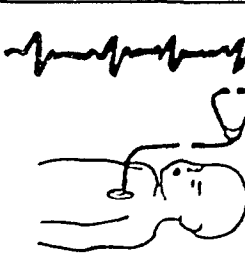
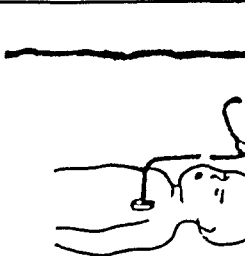
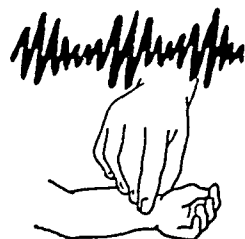
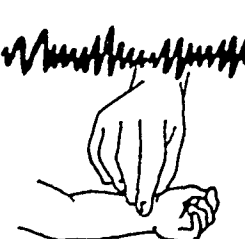
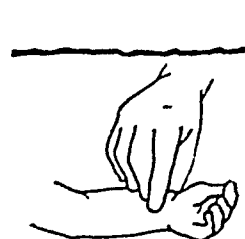
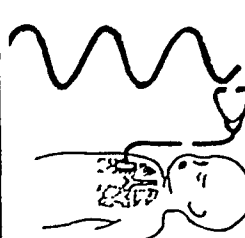
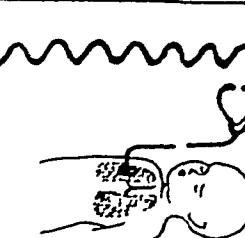
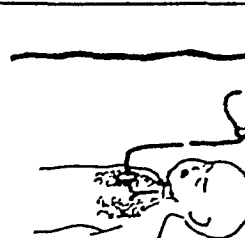
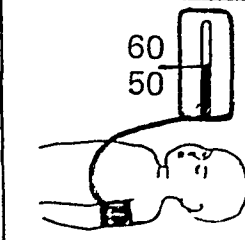
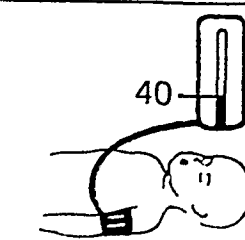
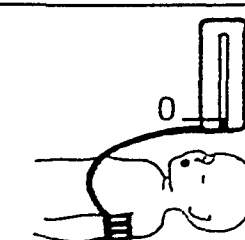

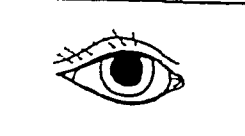
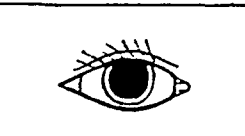
|                         | Preagony  | Agony   |   | Clinical death |
|-------------------------|---|---|---|----------------|
| Consciousness           | present   | confused  | absent  | absent         |
| heart sounds and ECG    |    |    |    |                |
| Pulse                   |    |    |    |                |
| Respiration             |   |   |   |                |
| Arterial pressure mm Hg |  |  |  |                |
| pupil                   |  |  |  |                |

Fig. 30 Terminal states (diagram)

substances, inadequate respiration and circulation exhaust the compensatory mechanisms and cause cessation of respiration and heart action

*Clinical death* This is the transitory state between life and actual death, during which irreversible changes do not yet take place in the most vulnerable tissues (first of all in the central nervous system) This accounts for the possible revival The onset of clinical death is characterized by the following signs absence of consciousness, respiration or blood circulation, complete areflexia and maximum dilation of the pupils The length of clinical death depends on the time during which the cerebral cortex can survive without blood circulation and respiration, and also on the following factors 1—the condition of the patient in the state of clinical death, 2—the character and duration of the period preceding death, 3—the cause of heart arrest, 4—age, and 5—body temperature

Terminal state, and clinical death in particular, are characterized by drastic disorders occurring in all vital functions the content of sugar, glycogen and macroergic phosphorus compounds (ATP, ADP) drops sharply, lactic acid content increases, the content of easily hydrolysed amide groups of proteins and conjugated amide groups decreases, and the ammonia concentration increases The cells of the cerebral cortex and the cerebellum are most vulnerable, the cerebral trunk is affected much less significantly Automaticity function and heart conduction are preserved for 20-30 minutes after the onset of clinical death The renal function is seriously upset due to decreased renal blood flow, active and passive reabsorption and secretion in the tubules are inhibited Vast affections of the renal and hepatic parenchyma may develop

The terms clinical death and terminal state can be associated with the term cardiac arrest and cessation of respiration, and vice versa Cessation or inhibition of any other function with preserved respiration and circulation of blood do not yet indicate the onset of terminal state or clinical death

*Aetiology* Causes of terminal state are quite varied Respiration can be stopped in children in severe injuries, burns, suffocation (drowning), carbon monoxide poisoning, poisoning with chemical substances, toxicoses, and inflammatory diseases (confluent pneumonia, sepsis, and the like), inhalation of inert gases (helium), nitrous oxide, etc The most frequent cause of cessation of respiration and asphyxia in children is obstruction of the airways of various aetiology due to some anatomical and physiological properties of the respiratory system (see Chapter 3)

Blood circulation is usually discontinued in children due to asphyxia, oxygen deficit, heart diseases, injuries, side-effects of medicinal preparations which act mostly on the heart, directly on the myocardium (adrenaline, procaine, novocainamid, cardiac glycosides, eu-

phylline) Heart can stop due to overdosage of narcotic substances, water-electrolyte disbalance, e.g., rapid injection of potassium preparations, and hyperthermia. The heart stops by reflex in children more often than in adults, it occurs due to insufficient anaesthesia during manipulations on the reflexogenic zones, e.g. the solar plexus, the lung root, during incision of the skin. Hypovolaemia, loss of blood or plasma, is especially dangerous in children. Insignificant loss of blood can result in marked hypovolaemia and cessation of blood circulation.

*Pathogenesis* The mechanism of cessation of respiration can be explained by the disturbed oxygen supply to the lungs due to the mentioned causes. Severe hypoxaemia, hypoxia, hypercapnia, and metabolic acidosis inhibit the respiratory centre. The mechanism by which the heart stops is varied and complicated. For example, overdosage of anaesthetics inhibits directly the conduction system of the heart. Anaesthetics block the respiratory link in the Krebs' cycle, inhibit the production of ATP, and decrease the normal function of the cell and its sensitivity. The reflex arrest of the heart is usually the result of strengthening reflexes of the *nervus vagus*, especially in the presence of hypercapnia. Marked disorders in potassium metabolism cause cardiac arrest because potassium is involved in the carbohydrate metabolism and it thus has its effect on contractions of the muscular fibres. In many cases the heart stops because of combination of hypoxia, hypercapnia, metabolic acidosis, and hyperkalaemia, which upset the excitability, conduction and contractility of the heart muscle.

The heart stops in various intoxications due to upset metabolism, which disturbs energy formation and conversion of chemical energy of the myocardium into mechanical energy.

*Clinical picture and diagnosis* The clinical picture of terminal state and clinical death is characterized by a full cessation of respiration and blood circulation, by complete absence of either of these functions, or their marked depression. The signs of marked *depression or absence of respiration* are cyanosis of the skin and the lips, complete absence of respiration or the presence of only slightest respiratory excursions, which are performed by accessory muscles. The absence of respiration is difficult to establish by mere observation of the chest. The only reliable sign of lung ventilation is exhalation. It can be detected by placing the bell of a phonendoscope (without membrane) or the ear to the mouth or nose of the patient.

*Cessation of blood circulation* often begins with prodromal signs 1—a pronounced fall of pressure, 2—bradycardia or marked tachycardia attended by circulatory disorders and arrhythmias. 3—changes in the skin and mucosa colour (rapidly developing cyanosis, greyish colour of the skin), 4—irregular respiration, 5—appearance of ven-

tricular extrasystoles, ventricular tachycardia, atrioventricular block of the 2nd and 3rd degree (ECG)

A sudden arrest of the heart is characterized by the following 1—absence of pulse in large arteries (the common carotid and femoral artery); 2—cessation of respiration, 3—absence of consciousness, 4—absence of heart sounds, 5—dilation of the pupils, 6—pallor and cyanosis of the skin

The absence of pulse on large arteries is the earliest sign of stopped blood circulation. Dilation of the pupils is a relatively late symptom of brain hypoxia. It develops 30-60 seconds after a sudden stoppage of blood circulation, this symptom should not therefore be waited for. After the arrest of the heart the skin is usually ash-grey. Cyanosis develops if hypoxia is the cause of stopped blood circulation.

### MANAGEMENT OF TERMINAL STATE RESUSCITATION

Terminal state and clinical death discontinue all vital functions of the body. The decay of the central nervous system is most dangerous. It has been shown experimentally and established clinically that the cells of the cerebral cortex can live no longer than 3-4 minutes in the absence of oxygen. Clinical death can sometimes last longer in the presence of hypothermia when the oxygen demand of the cells is low.

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**REMEMBER** 3 or 4 minutes is the time reserve within which a child with stopped blood circulation and respiration can be revived

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At present the only means of saving the cerebral cortex from decay in conditions of hypoxia are artificial lung ventilation, gas exchange and circulation of blood.

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The main requirement of resuscitation is maintaining artificial ventilation of the lungs and cardiac massage (artificial circulation of blood)

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Since the time during which successful treatment of children in the state of clinical death is very short, resuscitation measures should be started by the person who is the first to arrive at the site of accident. This person may or may not have any medical education or skills. Resuscitation measures are conducted in the absence of spontaneous respiration and blood circulation. But if a victim is in the terminal state, preagonal or agonal condition (with the signs of marked inhibition of respiration and blood circulation),



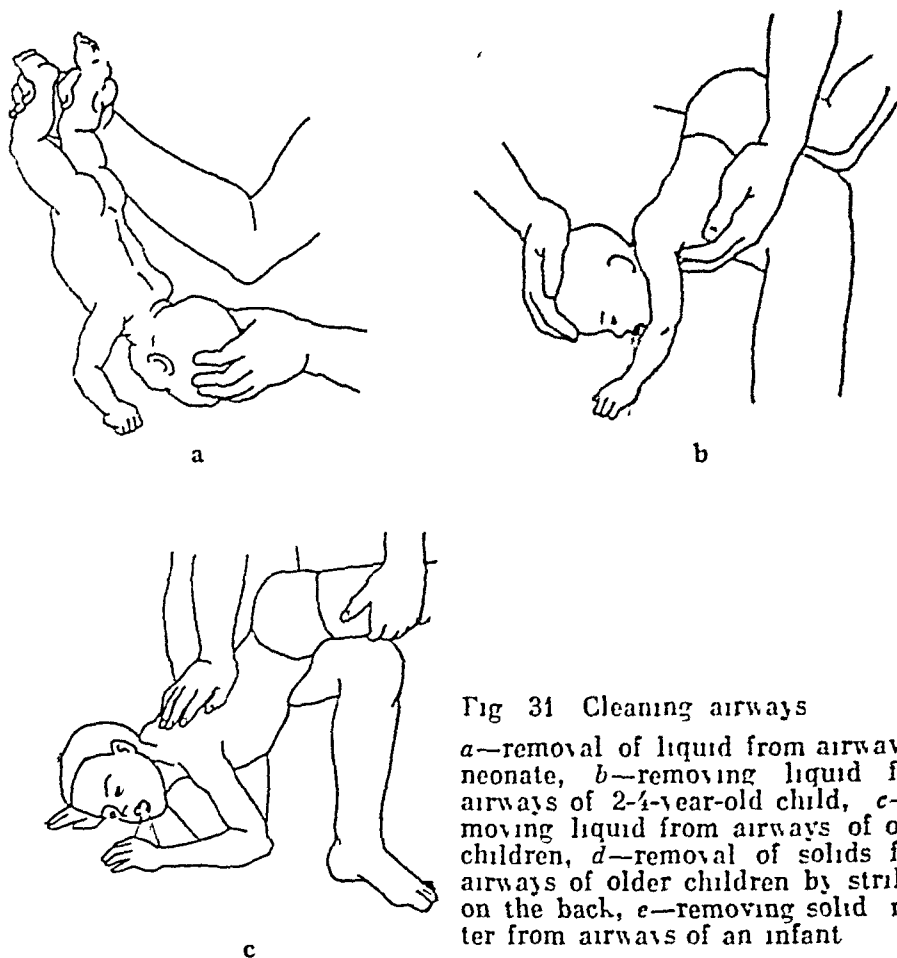


Fig 31 Cleaning airways

*a*—removal of liquid from airways of neonate, *b*—removing liquid from airways of 2-4-year-old child, *c*—removing liquid from airways of older children, *d*—removal of solids from airways of older children by striking on the back, *e*—removing solid matter from airways of an infant

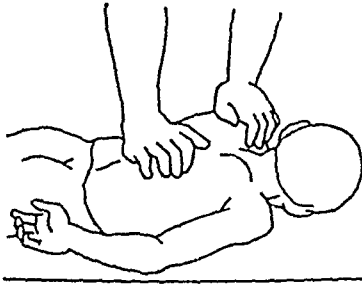
resuscitation measures should be taken without waiting for complete cessation of blood circulation or respiration

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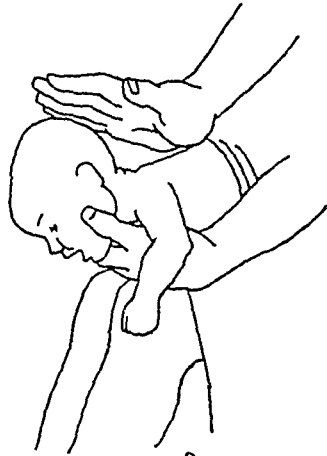
Do not waste time to establish an absolutely correct diagnosis of stopped respiration or blood circulation. Do not wait for ECG proofs. Resuscitation measures should be started at the moment when clinical death is suspected.

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Cardiopulmonary resuscitation can conventionally be divided into two stages. The first stage (it is sometimes called premedical) is the re-establishment of patency of the airways, artificial ventilation of the lungs, and closed chest cardiac massage. The second stage is a qualified medical aid including measures aimed at restoration of spontaneous circulation of blood and respiration (medicamentous therapy, electric stimulation of the heart, defibrillation) and post-



d



e

resuscitation management of children Re-establishment of the heart and lung action is sometimes called primary or direct resuscitation This can be shown as follows

1 Mark the time of cardiac arrest Give no medicines do not waste time!

2 Clean the victim's airways to ensure their patency

3 Place the child on his back over a rigid support (floor, table, etc)

4 Perform 2 or 3 artificial inhalations (mouth-to-mouth, from an air bag, or from the mask of the apparatus for anaesthesia)

5 Start closed chest cardiac massage (apply pressure to the chest 4 or 5 times)

6 Continue artificial ventilation of the lungs either by the mouth-to-mouth technique or by the mask Continue cardiac massage four pressure strokes per each inhalation

7 Continue artificial ventilation of the lungs by the mouth-to-mouth method or by the mask, intubate the trachea if possible and if necessary Continue closed chest cardiac massage Administer adrenaline, calcium chloride and sodium bicarbonate intracardially or intravenously

8 Place ice on the head and the large vessels

9 Start an electrocardiograph

10 Clean the tracheobronchial tree by aspiration, continue artificial lung ventilation If possible, use a respirator to supply a high-oxygen gas mixture If closed chest cardiac massage fails to give results in 2-3 minutes, perform a direct massage on the open heart Give another injection of epinephrine, calcium chloride and sodium bicarbonate

11 Continue artificial respiration If necessary, apply a defibrillator

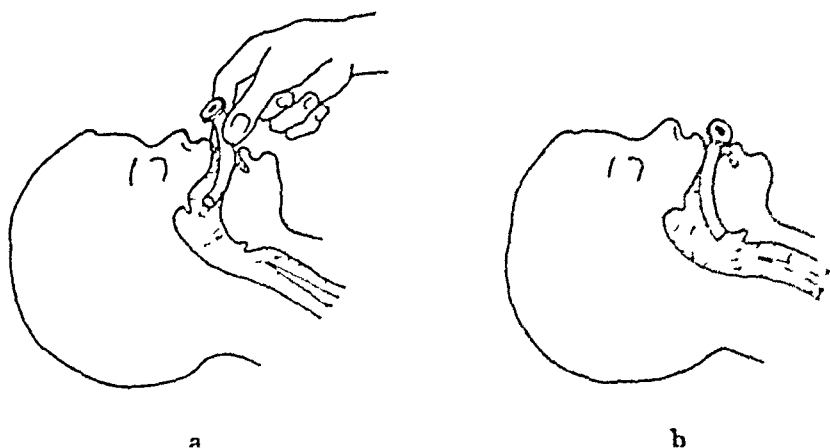


Fig 32 Introducing an airway by stages (a) and (b)

12 Continue artificial respiration Perform tracheostomy, if necessary Continue cardiac massage If much blood is lost, transfuse blood intravenously and intra-arterially

As can be seen, the resuscitation measures should be performed in a strict order Artificial ventilation of the lungs and cardiac massage should be performed simultaneously, or to be more exact, alternately

The airways should be cleaned by various methods, depending on circumstances (Fig 31) If the airways contain insignificant amounts of extraneous matter, the following should be done a—the child is placed in supine position over a rigid support (table or the like), b—the child's head is pulled back sharply, c—his lower jaw is pulled forward, d—the mouth and the pharynx are cleaned using a tampon, e—an airway is passed into the mouth, f—if necessary, the trachea is intubated and the tracheobronchial contents removed by aspiration

Much liquid can be removed from the airways of a neonate or a nursingling by holding him by the legs with the head down The child unbends slightly his head in this position and the extraneous matter can then be removed from his mouth using a finger If a child is older than one year, he may be placed on the resuscitator's lap and his head lowered down

When resuscitation measures are being taken, the child should be placed in a horizontal position (except certain moments when it is necessary to free his airways from large amounts of fluids). The child's head should preferably be slightly lowered and turned on its side A rest should be placed under the shoulders so that the child's head is tilted back Extreme backward position of the head or its resting against the chest can narrow the airways due to retraction of the tongue root

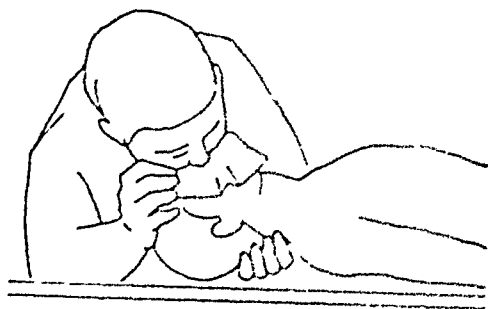
Deflexion of the head is a simple manipulation. It ensures free patency of the airways by stretching tissues located between the larynx and the mandible. The tongue root is thus moved away from the posterior wall of the pharynx. Maximum deflexion of the head opens the airways in 80 per cent of unconscious patients. The head of a child should be tilted back with two hands. The resuscitator should stand at the head of the child or by his side. One hand should be placed under the patient's neck, while the other hand is on the child's forehead to deflect the head maximum. The head of infants may be deflected by one hand only by pressing the mandible (at the chin) backward and upward (with respect to the patient's position).

The mandible of an unconscious patient should be pulled forward. This can be done in three stages: a—the mandible is lowered by pressing the chin below the lip by both thumbs, the 2nd and 3rd fingers of both hands are placed on the angles of the mandible, b—the mandible is pulled by the 2nd and 3rd fingers anteriorly to displace the lower teeth with respect to the upper ones, c—the mandible is lifted and held in this position by the 2nd and 3rd fingers during the entire procedure.

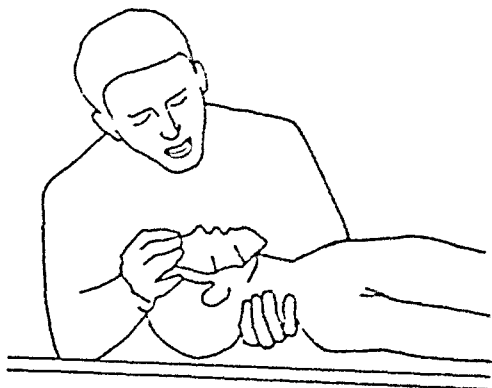
In order to keep the tongue in the desired (forward) position, airways (Fig 32) are used. As the airway is passed into the mouth, it is not necessary to hold the mandible any longer, which facilitates significantly the further resuscitation measures. In case of trismus, the mouth can be opened by introducing the 2nd finger between the last molars. In some cases it is necessary to pull the tongue of infants and fix it with tongue holder.

The resuscitation measures are mainly aimed at maintaining circulation of blood with the minimum possible arterial pressure (50-60 mm Hg) and at ensuring effective alveolar ventilation of the lungs. The lungs may be ventilated by the exhaled air, atmospheric air, or, which is better, by oxygen or an air-oxygen mixture.

**Mouth-to-mouth or mouth-to-nose artificial respiration.** These techniques require no special apparatus, which is their main advantage. The procedure is performed as follows: the head of the victim is deflected as much as possible by the resuscitator, who presses his mouth to the mouth of the child and forcefully blows air into his lungs (the nostrils of the child should be pinched during this operation). The head of the patient should then be moved aside and the resuscitator must be sure that the child's chest lowers (Fig 33). If air passes into the stomach, it remains there, and the upper abdomen rises with each next artificial inhalation. The air pressure in the stomach displaces its contents into the airways. If the patient's head is pulled back, the airways become patent and less air passes into the stomach. If a gastric tube is not in the stomach, or if the resuscitator is not sure that the child's stomach is empty, it



a

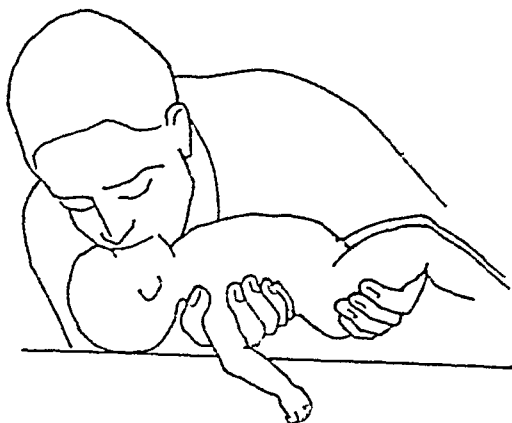


b

Fig 33 Artificial respiration (mouth-to-mouth method)

a—blowing air into patient's lungs,  
b—exhalation

tion of gas exchange and causes  
piratory centre during each distension



is better not to press on the stomach because its contents may get into the airways.

Artificial mouth-to-mouth or mouth-to-nose respiration should be performed at a rate of 40 cycles per minute for a neonate and 20-24 for older children. When resuscitating neonates and nurslings, the force of blowing should be softer in order to prevent possible damage to the airways and lungs of the child (Fig 24). The criterion of efficiency of artificial respiration is sufficient movement of the chest and the diaphragm and lessening or disappearance of signs of hypoxia. If the chest does not rise in response to blown air, the airways should be cleaned, or the resuscitator should press his mouth more closely to the mouth and/or nose of the victim to prevent leakage.

Mouth-to-mouth and mouth-to-nose artificial respiration are the most effective methods since air is blown directly into the lungs. This promotes normalization of reflex stimulation of the respiratory tissue. Moreover, mouth-to-mouth artificial respiration has the following advantages over other methods: the hands of the resuscitator

Fig 34 Artificial respiration given to a newborn by the mouth-to-mouth-and-nose method

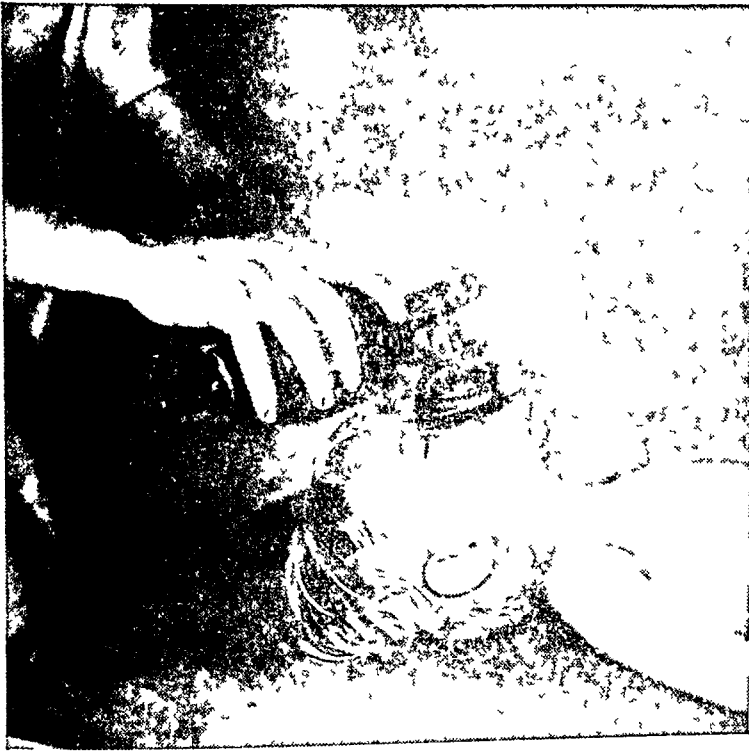


Fig 35 Artificial lung ventilation by a face mask and an air bag

remain free and he can thus feel the movements of the chest and discover possible obstruction of the airways

Every medical worker must be able to perform the mouth-to-mouth artificial respiration. Health education of population should also include training in giving artificial respiration by this method.

S-tubes, masks and air bags should be used for convenience of giving artificial respiration. An apparatus shown in Fig 35 is widely used for artificial ventilation of the lungs. The air bag has a valve which closes when pressure is applied and the air contained in the bag is thus expressed into the patient. When giving artificial respiration using this apparatus, the mandible should be pulled forward and the mask applied tightly to the child's face. The head should be deflected and the air bag pressed until the chest expands. The mask should then be removed so that the patient can exhale. The cycle should then be repeated. In order to ensure a tight contact between the mask and the face of the patient, the part of the mask over the nose should be pressed by the thumbs, while the mask part overlying the chin, by the index fingers. The other fingers should be

used to pull the child's chin upwards and back. Noisy respiration indicates obstruction of the airways, which may be due to retraction of the tongue root or accumulation of sputum. The respirator with an air bag can be used to supply an air-oxygen mixture for artificial ventilation of the lungs. In an ambulance car or in the hospital, the child is given artificial respiration from a special apparatus.

**Artificial circulation of blood.** When blood circulation discontinues, injections of medicinal substances—intravenous or intra-arterial infusions give no effect. The only means by which circulation of blood can be maintained artificially is cardiac massage. When a cardiac arrest is suspected, closed chest massage should be started immediately (Plate 4). The advantages of indirect (closed chest) cardiac massage are that it can be performed by non-medical personnel, that it does not require any special conditions, and that no time is necessary for opening the chest for direct massage.

**Closed chest cardiac massage.** The child is placed on a rigid support. If the resuscitating brigade includes two persons, one performs cardiac massage while the other artificial respiration. When performing cardiac massage on an older child, the resuscitator places the heel of one hand on the lower third of the sternum and the heel of the other hand on top of the first (Fig. 36). Pressure should be applied to the chest at regular intervals, at a rate of about 70-80 per minute, which corresponds approximately to the heart rate of a child of this age. In order to prevent fractures of the ribs, pressure should not be applied to the xiphoid or the sides of the chest. Both thumbs (Fig. 37b) or two fingers (Fig. 37a) should be used to apply pressure to the chest of infants or neonates. The resuscitator puts one hand under the infant's back to support the left side of his chest and uses one or two finger-tips of the other hand to apply rhythmic pressure to the lower end of the sternum, just above the xiphoid. The pressure exerted should move the sternum 1.5-2 cm toward the spinal column and ensure a distinct artificial pulse wave on the carotid or femoral artery. The rate at which the pressure is applied to the infant chest should be 100-120 per minute.

If indirect cardiac massage is effective, it manifests by the following signs: 1—appearance of pulse on the carotid and radial arteries, 2—elevation of the arterial pressure to 50-70 mm Hg, 3—disappearance of cyanosis or pallor and reddening of the skin, 4—narrowing of the pupils and their reaction to light, the eye-balls begin moving, 5—appearance of spontaneous respiration.

The absence of these signs during the course of 2-3 minutes indicates the necessity of open chest cardiac massage in hospital conditions. Outside hospital or in polyclinic conditions, or at clinics other than surgical, closed chest cardiac massage should be carried out for at least 10-15 minutes. Indirect cardiac massage is often effec-

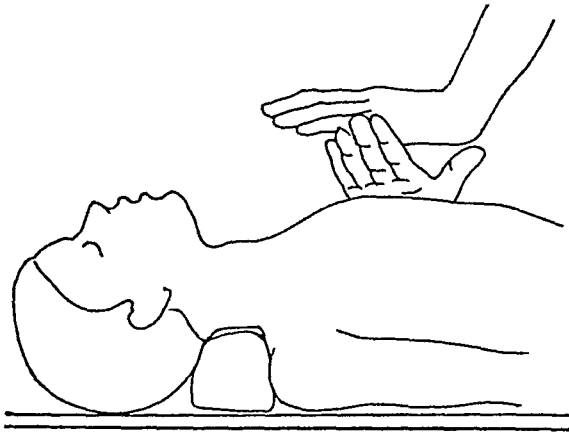


Fig 36 Closed chest cardiac massage with two hands

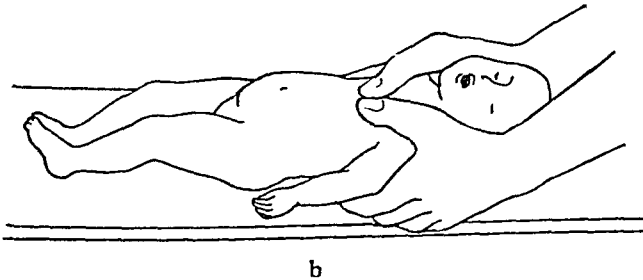
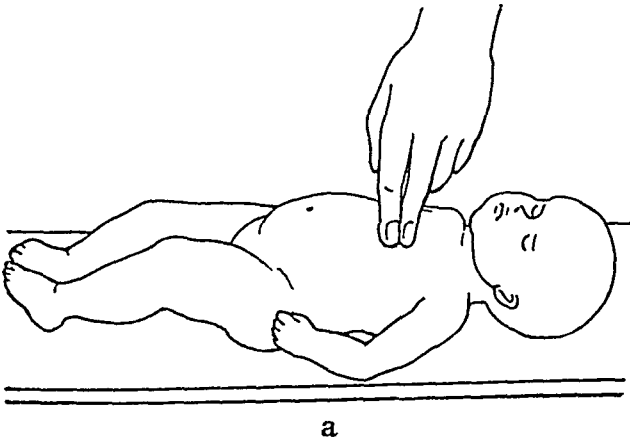


Fig 37 Closed chest cardiac massage with (a) two fingers, (b) two thumbs

tive with infants because their chest yields easily to the pressure of the hand. But if conditions permit, ineffective indirect cardiac massage should be discontinued in 90-120 seconds in the presence of profuse bleeding, severe intoxication or myocarditis. Direct massage of the heart in the opened chest should be performed instead



Relative contraindications to closed chest cardiac massage are (a) funnel-shaped chest, (b) fracture of several ribs, (c) bilateral pneumothorax, (d) cardiac tamponade, and (e) cardiac arrest in systole

Complications of closed chest cardiac massage are fractures of the ribs or sternum, pneumo- and haemothorax

**Combined use of cardiac massage and artificial respiration.** Closed chest cardiac massage should be performed simultaneously with artificial ventilation of the lungs. These two operations should be alternated at a rate of 4 : 1, i.e. one artificial inhalation should be followed by four pressure strokes

**Medicamentous help during resuscitation** Medicinal substances are administered only after cardiac massage is started. Epinephrine or norepinephrine should be administered in a dose of 0.25 mg to neonates and to 0.5 mg to older infants and children (the degree of dilution is 1 : 1000). Calcium chloride (2.5 ml of a 5 per cent solution) should be injected together with adrenaline or by a separate injection. When the heart is compressed, blood is ejected from the left ventricle into the greater circulation and the heart vessels. If the tone of the heart muscle fails to be restored, another adrenaline injection is required. The calcium ion promotes conversion of energy into mechanical contraction of the muscle fibres. Low calcium concentration in the plasma and decreased intracellular calcium account for decreased systolic force of the muscle and dilatation of the heart. In addition to adrenaline and calcium chloride, 1.5-2 ml/kg of a 8 per cent sodium bicarbonate solution is also injected through the same needle. After the heart has started beating again, 40 ml of a 20 per cent glucose solution with insulin and vitamins are injected intravenously to normalize myocardial metabolism

**Puncture of the left ventricle** A 6-8 cm long needle is used to puncture the heart. The needle should pass perpendicularly to the surface of the sternum, by its left side, in the 4th or 5th intercostal space, by the upper edge of the lower rib. As the resilient muscle of the left ventricle is being punctured, the physician feels a slight resistance. The appearance of blood in the syringe (either spontaneously or when the syringe piston is pulled back slightly) indicates that the needle is inside the ventricle cavity. Care should be exerted that no air is present in the syringe during intracardiac injections. The aorta can occasionally be punctured if the needle deviates from the right course. No blood appears as the piston of the syringe is pulled back again. Calcium chloride should not then be injected because the position of the needle is unknown. The puncture should then be repeated.

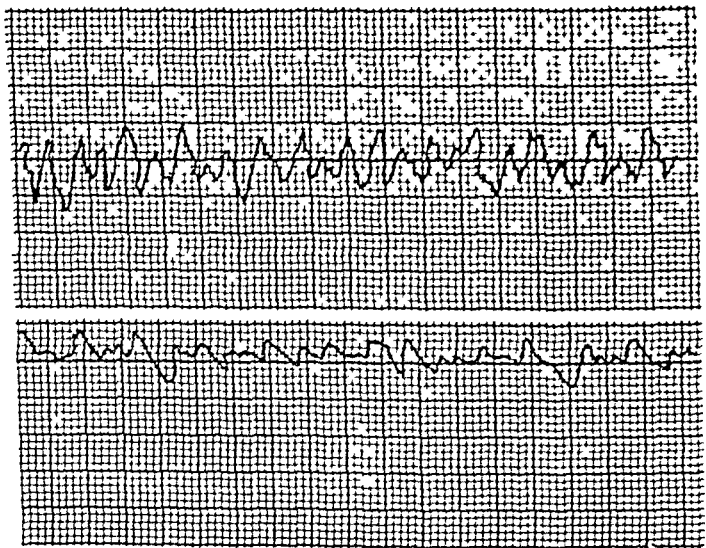
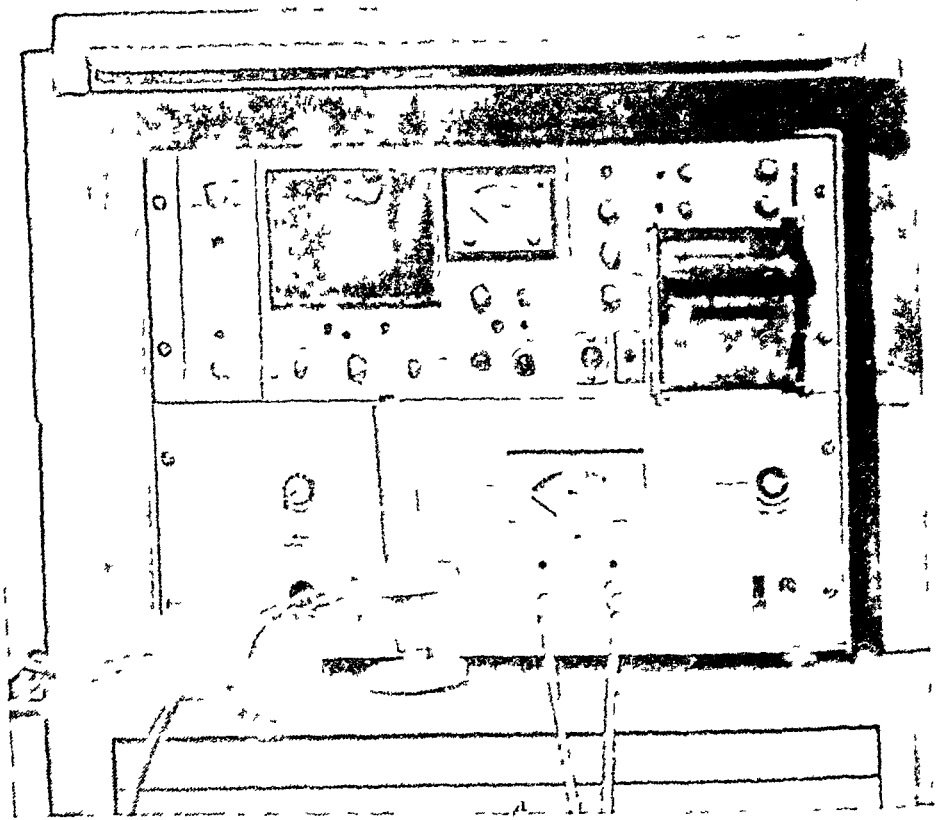
If the subclavian vein is catheterized, medicinal preparations can be infused without cardiac puncture.

**Open chest cardiac massage** The direct cardiac massage is indicated in the following cases: 1—the heart stops during operation on the chest; 2—ineffective closed chest cardiac massage, 3—closed chest cardiac massage is contraindicated. The chest is opened by the 4th intercostal space on the left side, at a distance of 1.5–2 cm from the sternum edge to the midaxillary line (Plate 5). Massage of the heart begins after opening the chest and the pleura. The heart of neonates and infants under 1 year of age should be best pressed against the posterior surface of the chest by two fingers. Opening the pericardial sac is obligatory only if liquid is present in it. The heart of older children should be pressed by the right hand, with the thumb placed over the right ventricle and the other fingers and the palm—over the left ventricle. The heart should be compressed with the fingers held tightly together to prevent perforation of the heart muscle. The rate, at which the heart is compressed, depends on age and varies from 100–120 to 70–80 per minute. Adrenaline, calcium chloride and sodium bicarbonate are administered into the left ventricle. Complications of direct cardiac massage are incision of the internal thoracic artery and injury to the heart muscle (rupture, haemorrhage).

If the heart suddenly stops during operation on the abdominal organs, the heart can be given a direct massage through the diaphragm (Plate 6).

**Fibrillation of the heart** A severe complication of resuscitation is ventricular fibrillation. Fibrillar contractions of separate muscular fibres of the ventricles are the result of random continuous excitation due to slowed conductivity in the heart conduction system. Ventricular fibrillation occurs in hypoxaemia, hypercapnia, hyperkalaemia, haemorrhage, irritation by anaesthetics, excessive irritability of the myocardium. Ventricular fibrillation is diagnosed either by ECG (Fig 38b) or by direct visualization, if the chest is open. The heart should be defibrillated by an electric discharge (Fig 39).

**Indications for resuscitation** Indications and contraindications for resuscitation is a difficult problem. On the one hand, even the least hope for revival should be used by medical workers as an indication to resuscitate all children in the state of clinical death. But on the other hand, it is quite evident that resuscitation is reasonable only in cases where there is a hope to re-establish all vital functions of the body and to return the child to normal life. Hence the question, if it is really reasonable to resuscitate children with severe and incurable diseases, especially so if the cerebral cortex has decayed due to hypoxia during clinical death. What are the criteria for brain death? What are the indications for discontinuation of further attempts to revive a child? Opinions of researchers with respect to importance of separate clinical signs for prognosis of resuscitation are controversial. Experience shows that the absence



b

Fig 38 Defibrillator (a) and ECG during fibrillation (b)



Fig 39 Defibrillation

of consciousness for more than 12 hours is a bad prognostic sign. Electrophysiologists maintain that the absence of bioelectrical activity of the brain (level EEG) for 2-6 hours indicates death of the cerebral cortex, although cases were reported where patients recovered after electroencephalographic 'silence' for longer periods of time. Unanimity is absent in assessing such prognostic signs as the time of re-establishment of blood circulation, spontaneous respiration, or reflexes.

The cold pressor test, intravenous administration of atropine, bemegride, determining  $PO_2$  in the in- and outflowing blood, angiographic and computerized tests are used as criteria of brain death.

Cold water ( $3-4^{\circ}C$ ) is poured into the ear and the vestibulo-ocular reflex is determined: the eye-balls remain motionless if the brain is dead. ECG is taken or monitoring is performed during intravenous injection of a triple dose of atropine. The absence of response of the heart to this injection indicates brain death. Bemegride is a powerful respiratory analeptic. It stimulates the central nervous system in patients in a comatose state. Bemegride is administered intravenously with EEG control. The absence of changes in the EEG indicates brain death.

Oxygen tension in the blood is determined by puncturing the carotid and the internal jugular vein. The arteriovenous difference of oxygen in the inflowing and outflowing blood is normally 6-7 per cent. This difference drops to 0-1 percent in brain death. Angiographic and radioisotopic studies determine the cerebral blood circulation. The absence of such circulation indicates brain death.

When deciding whether or not resuscitation measures should be taken, a combination of various clinical signs and electrophysiological findings should be considered. The absence of consciousness or of sensitivity to pain, the absence of reflexes (pupillary, tendon reflexes) or muscular tone, of spontaneous respiration and bioelectrical activity of the brain during 2-3 hours indicate that the cerebral cortex is dead and further resuscitation measures are useless.

*Indications* Resuscitation is indicated for all children (except in the presence of contraindications which follow below) in the state of clinical death, agony or preagonal state.

*Contraindications* Resuscitation measures are not indicated in the following cases: 1—to children in the terminal stage of incurable diseases, 2—to children with malignant newgrowths and metastases, 3—to children with severe irreversible diseases and brain affections.

Resuscitation measures should be discontinued in the presence of a complex of signs of irreversible changes in the brain, such as (a) the absence of consciousness and biological activity (level EEG) for 24 hours, (b) permanent dilatation of the pupils, the absence of pupillary response to light, and the absence of the corneal reflex, (c) muscular hypotonia, (d) absence of adequate circulation of blood, prolonged hypotension (below 30 mm Hg), the absence of the ECG complex and absence of bleeding from an injured artery. The decision concerning discontinuation of resuscitation measures should be taken by the physician, anaesthesiologist-resuscitator, the head of the department and of the particular medical institution (Plate 7). The opinion of the neuropathologist is desirable as well.

### INTENSIVE THERAPY AFTER RESUSCITATION

Hypoxia of tissues, metabolic acidosis and severe intoxication with underoxidized substances persist for a long time in revived patients. Nowadays general principles of treatment of patients during post-resuscitation period have been worked out on the basis of the available clinical data. These principles are as follows: (1) elimination of hypoxia, (2) correction of acid-base balance, (3) correction of water-electrolyte metabolism, (4) prevention and treatment of dysfunction of vital organs and systems of the body, such as the brain, kidneys, liver, cardiovascular and respiratory systems.

Hypoxia can be removed by artificial ventilation of the lungs. A common mistake is undue hastiness in discontinuation of artificial lung ventilation, which is necessary even after a transient arrest of the heart and respiration. The criterion for discontinuation of artificial ventilation is  $PO_2$  not below 70 mm Hg during spontaneous respiration (without assistance of the external respirator). Accelerated respiration, retraction of the yielding parts of the chest and a fall of  $PO_2$  below 70 mm Hg are indications for continuation of artificial respiration. Hypoxia of tissues can also be lessened in conditions of hyperbaric oxygenation, which should better be conducted at a pressure of 3 atm. Children after clinical death should be given 3-4 sessions a day. A pathogenetically substantiated treatment of disordered acid-base balance is elimination of hypovolaemia and hypotension, and improving microcirculation in the peripheral tissues. Acid-base balance should be corrected during the post-resuscitation period only after determining its main indices.

Balancing water-electrolyte metabolism during the post-resuscitation period is difficult and requires complex therapy during many days. When prescribing infusion therapy, the daily demands and pathological losses of fluid should be determined. The excretory function of the kidneys should thoroughly be controlled. The selection of solutions for infusion therapy depends on the type of disorder of the water-electrolyte metabolism. The osmotic and oncotic pressures are obligatory measured.

To prevent hypoxic oedema of the brain, big doses of hormones (10 mg/kg hydrocortisone, 2-3 mg/kg prednisolone) are given in addition to artificial lung ventilation. Since it is the brain that is mostly damaged during clinical death, measures are taken immediately during early post-resuscitation period to protect the brain from hypoxia. Energy demands of the brain are reduced, intracellular metabolism is normalized, cytoplasmic membranes are restored, cerebral circulation is normalized, and oedema of the brain prevented. Sodium oxybate and barbiturates are now used to decrease the energy demand of the brain. If consciousness is not regained within 5 minutes after re-establishment of cardiac activity and respiration, big doses of thiopental sodium (to 30 mg/kg) should be administered intravenously at a maximum possible rate (depending on the stability of the haemodynamic indices). Thiopental sodium should be administered as early as possible, not later than 2 hours after re-establishment of blood circulation. From 10 to 20 mg/kg of the 2 per cent solution should be administered during the first 10-30 minutes, while the remaining dose should be administered as a 0.4 per cent solution during the course of 6 hours of the post-resuscitation period. The mechanism of thiopental sodium action is unknown. In the opinion of some authors, it decreases metabolism of the brain, improves blood circulation in the ischaemia-affected por-

tions, decreases intracranial pressure and intra- and extracellular oedema, stabilizes membranes, and has anticonvulsive and sedative effect

The patient should be given preparations improving brain metabolism (ATP, ascorbic acid, vitamins B, insulin with glucose, glutamic acid) During the first day of the post-resuscitation period (after re-establishment of blood circulation), nootropil (pyracetam) and aminalton should be administered Cerebrolysin should be administered in a week (1 ml every other day) Dehydration therapy during post-resuscitation period should be conducted only in the presence of brain oedema and elevated intracranial pressure Syndromes should also be treated

### RESUSCITATION OF NEONATES

The most common causes of neonatal death during the first hours and days following birth are hypoxia of the foetus and asphyxia due to strangulation of the neonate by the umbilical cord entangled round the neck or premature placental detachment Oxygen and carbon dioxide exchange through the placenta is thus disturbed Acute hypoxia can also be the result of pathological delivery with acute obstruction of the blood flow to the foetus during protracted or convulsive contractions of the uterus, prolonged standing of the foetal head at the pelvic inlet after premature escape of the fluids, or contracted pelvis, uterine rupture, acute hypoxia of the mother associated with blood loss

The absence of respiration or inhibited spontaneous respiration of a neonate can be caused by the labour injury (derangement of cerebral circulation, intracranial haemorrhage) and obstruction of the airways due to aspiration of the amniotic fluid, meconium, blood, mucus, etc

Neonatal asphyxia is often caused by pharmacological preparations that are administered to the mother during labour, which penetrate the placenta and cause damage to the foetus Morphine, promedol, barbiturates (thiopental sodium, hexenal), halothane, ether, stimulants of uterine contractions (oxytocin in big doses) easily pass the placental barrier In addition to these preparations, the foetus can also be damaged by local anaesthetics that are administered during labour (procaine, lidocaine, trimecaine)

Irrespective of the cause, the pathogenetic mechanisms of asphyxia are the following 1—collapsed lung, 2—non-compensated metabolic and respiratory acidosis, 3—inhibition of the respiratory centre and its insusceptibility to high  $PCO_2$

*Treatment* The amount of resuscitation measures and the length of intensive therapy in neonates born at term depend on the gravity of asphyxia and are assessed in points by the end of the 1st and 5th

minute (the Apgar scale) 6-5 points indicate slight asphyxia, 4-3 points indicate asphyxia of medium gravity, and 2 and less points indicate severe asphyxia. Neonates scoring 10-7 points by the end of the first minute after birth need no special resuscitation measures.

The following measures are necessary to take in slight asphyxia:

- 1 The neonate is placed on the table and his head is slightly deflexed

- 2 Mucus, fluids and blood are removed from his airways by aspiration to ensure their patency. A special rubber bulb or an electric aspirator is used for the purpose.

- 3 Oxygen is given to breathe by a mask. Assisted respiration or artificial lung ventilation with pure oxygen or helium and oxygen should be conducted if necessary.

- 4 A 4 per cent sodium bicarbonate solution is administered intravenously (4-8 ml depending on the body weight of the neonate).

- 5 A 10 per cent calcium gluconate (3 ml) is administered intravenously with 10 ml of a 20 per cent glucose solution, 1 ml of a 0.3 per cent aethimizol.

- 6 Cocarboxylase (50 mg) in 10 ml of haemodes (neocompensan) and 2-3 ml of a 2.4 per cent euphylline are administered intravenously.

- 7 The upper airways are cleaned.

- 8 Acid-base balance is determined and corrected if necessary.

- 9 If the respiratory centre is inhibited with narcotics, analeptics should be used (cordiamine, nalorphine, corazol, bemegride, etc.).

Neonates with asphyxia of medium gravity are treated as those with slight asphyxia, but if the signs of asphyxia intensify, the trachea should be intubated using a laryngoscope and the lungs should be ventilated artificially.

Resuscitation measures for neonates with severe forms of asphyxia are as follows:

- 1 The neonate is placed on the table and his head pulled back slightly.

- 2 Mucus, blood and fluids are removed from his oropharynx.

- 3 Artificial respiration is given using a portable respirator or an apparatus, pure oxygen should be used for the purpose. If spontaneous respiration is not established in 1 minute, the trachea should be intubated and liquids aspirated from the airways again. Artificial ventilation of the lungs should be performed through the tube. When spontaneous respiration is established, assisted respiration should be conducted.

- 4 In the absence of heart action or in the presence of marked bradycardia, closed chest cardiac massage should be conducted. If the heart action is not re-established within 1 minute, 0.2 ml of adrenaline with 1-2 ml of calcium chloride should be administered intracardially. Using the same needle, 8-10 ml of a 40 per cent



sodium bicarbonate solution is injected from another syringe. The needle is then removed and closed chest cardiac massage continued.

5 Bags with ice should be put round the child's head.

6 In order to protect the brain from hypoxia, sodium oxylate is injected slowly into the vein (100 mg/kg) in 10 ml of haemodes (neocompensan).

7 1 ml (10 mg) of lasix is injected intravenously.

8 If resuscitation is successful, the tracheobronchial tree is cleaned again, acid-base balance is assessed, and potassium and sodium content of the plasma and erythrocytes determined.

9 In order to prevent posthypoxic oedema of the brain and to treat posthypoxic affections of the central nervous system, craniocerebral hypothermia and hyperbaric oxygenation are indicated.

Respiration and cardiac action can in most cases be successfully re-established. But the study of late results has shown that irreversible changes occur in the central nervous system of some children; the resuscitator should therefore consider the circumstances to reason the length of resuscitation procedures. There are no criteria to determine the degree of affection of the central nervous system and its reversibility. The Apgar scoring is practically used everywhere to assess the general condition of neonates. If a neonate scores from 0 to 3 points, the resuscitation should not continue more than 10 minutes, if no appreciable improvement is attained during this time. If the condition improves and can be assessed as 4-7 points, resuscitation should continue for 2-3 hours.

The study of late results of resuscitation shows that if resuscitation measures are taken during the first 4 minutes of asphyxia, no pathological changes in the central nervous system occur in children in the early or late periods. If asphyxia lasts to 10 minutes, the neonate's condition normalizes by the 8-10th day. If asphyxia lasts for more than 10 minutes, the child develops convulsions, he has focal affections of the central nervous system and psychic disorders. These findings indicate the importance of early resuscitation measures.

## Chapter 17

### General Principles and Methods of Correcting Respiratory Insufficiency

*Aetiology and pathogenesis* Respiratory insufficiency in children depends on the following inter-related factors: disordered central regulation and muscular dysfunction, obstruction of the airways, rigidity of the chest and pulmonary tissue, upset intrapulmonary ventilation and perfusion. The following factors upset the central respiratory regulation: injury, disturbed cerebral circulation, oedema

of the brain, intoxication, residual or perverted effects of muscle relaxants, anaesthetics, and narcotic analgesics. Peripheral nerves and muscles are affected in myasthenia, polyneuritis, poliomyelitis, botulism, labour injury, and immaturity of the respiratory centre.

The airways become obstructed due to aspiration of amniotic fluid, fluids contained in the stomach and the oropharynx. This usually occurs in neonates with defects and diseases of the gastrointestinal tract. The airways can also be obstructed by foreign objects or sputum, air supply can also be upset due to obstruction or collapse of the endotracheal tube or tracheostomic cannula. Obstruction of the airways can result in oedema of the subglottic space of infectious or traumatic aetiology. The airways can also be obstructed due to inhibited function of ciliary epithelium and changes in the rheological properties of the sputum, e.g. during atropinization and in the presence of growth defects.

The chest becomes rigid in the presence of pneumothorax, haemothorax, diaphragmatic hernia, high diaphragmatic cupola in intestinal obstruction, peritonitis, and in ulceronecrotic enterocolitis. The elastic properties of the lungs are impaired during emphysema, pneumofibrosis, interstitial oedema, and decreased surfactant content in neonates with diseases of hyaline membranes, pneumonia, and hypoplasia of the lungs.

A very important factor responsible for respiratory insufficiency is the upset ratio of the pulmonary ventilation to perfusion. Non-uniform ventilation is especially pronounced in diseases of the lungs attended with contraction of the airways, e.g. in bronchial asthma, emphysema, bronchiolitis, tumours of the lung, and pneumonia. Prolonged motionless position of the patient during operation, anaesthetics and muscle relaxants intensify non-uniformity of lung ventilation.

Perfusion of the lungs depends on the volume of circulating blood, contractile power of myocardium and pulmonary vascular resistance. Either of these factors can disturb the gas exchange function of the lungs. The extreme degree of ventilation-perfusion disorders is the intrapulmonary shunt, i.e. the discharge of non-oxygenated blood into the arteries.

### INTENSIVE THERAPY OF RESPIRATORY INSUFFICIENCY

The methods of correcting respiratory insufficiency can be divided into several groups.

**Maintaining patency of airways.** Keeping the airways unobstructed is the most important measure in treating respiratory insufficiency in children. The simplest way of re-establishing patency of the airways is *maximum deflection of the child's head* in the atlanto-occipital articulation with simultaneous protrusion of the mandible. The

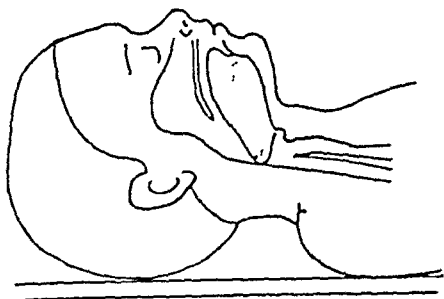


Fig 40 Obstruction of the airways by tongue root



Fig 41 Correct position of the head and mandible during artificial lung ventilation

tissues lying between the larynx and the mandible become strained and the tongue root separates from the posterior wall of the pharynx (Figs 40 and 41) In order to facilitate deflection of the head, a roll is placed under the child's shoulders This method is commonly used during emergency resuscitation or after administration of muscle relaxants before the tracheal intubation

But it is difficult to maintain free passage of air through the airways by this method for a long time *Oral or nasal artificial airways* are therefore used to prevent tongue retraction Airways are commonly

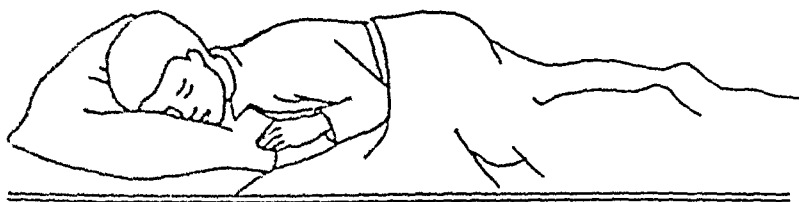
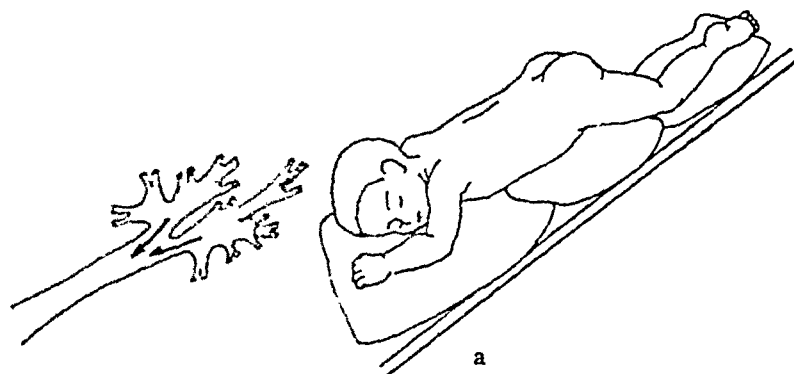
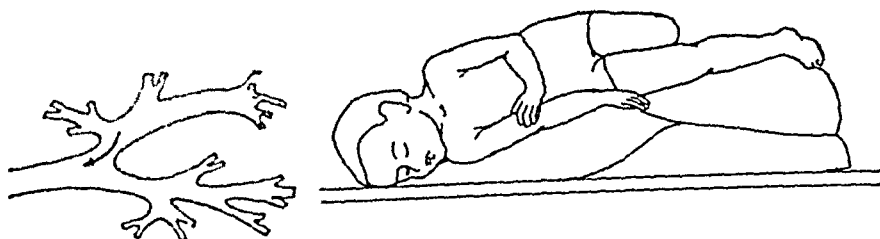


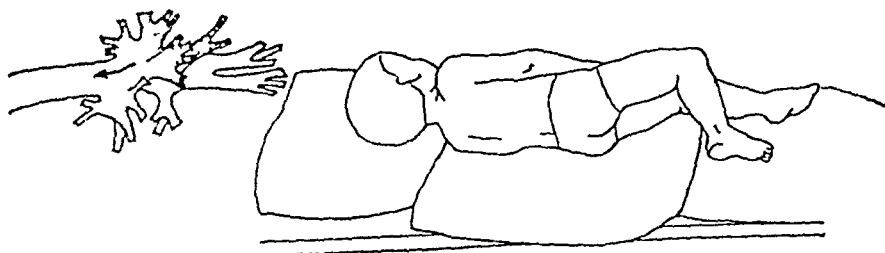
Fig 42. Post-operative position in bed



a



b



c

Fig 43 Positions facilitating drainage

a—for drainage of both bronchi, b—for drainage of the main left bronchus,  
c—for draining the main right bronchus

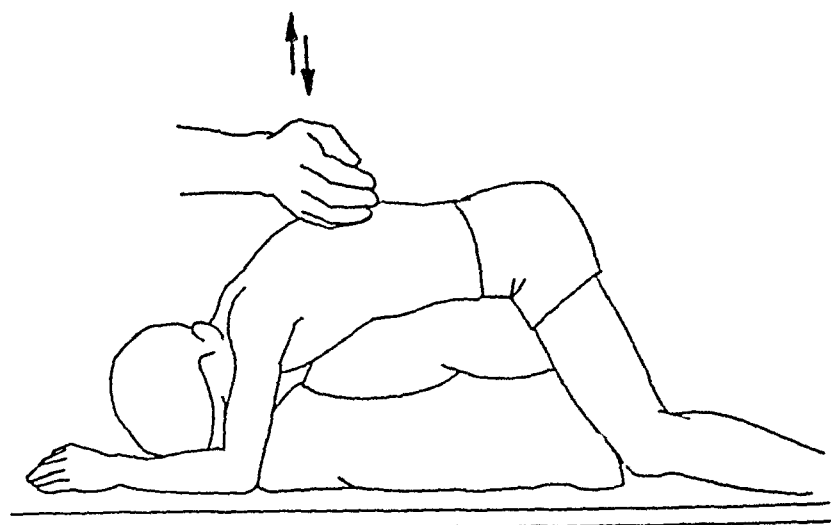


Fig 44 Percussion massage

used during resuscitation or during anaesthesia with spontaneous lung ventilation. The anaesthesiologist should remember that artificial airways often provoke vomiting.

The *child's position* in bed or couveuse is important for prevention of airways obstruction. After operation or anaesthesia the infant is usually placed horizontally on his side or in the prone position (Fig 42). The neonate should be kept in the vertical position for 10-15 minutes after feeding, this prevents occasional aspiration of the gastric contents. For the same reason infants susceptible to regurgitation should lie in bed or couveuse with their heads slightly elevated.

Ample viscous purulent mucus is accumulated in the tracheobronchial tree of infants with atelectasis, lung abscess or pneumonia. These patients should therefore be placed in the *drainage position* (postural drainage) from time to time. Fig 43 shows various positions in which conditions are provided for draining fluids from the airways. It is necessary to change the position of a child in his bed even if he does not discharge sputum, the ventilation-perfusion conditions in the lungs are thus improved and infectious complications prevented. Placing a child in a position facilitating drainage of fluids is usually combined with percussion massage, stimulation of the coughing reflex and inhalation therapy. *Percussion massage* is performed by compression and striking the chest at sites overlying cavities to be drained (Fig 44).

*Catheterization of the airways* for aspiration of their contents restores in most cases the patency of the tracheobronchial tree. The oral and nasopharyngeal cavities are usually catheterized without visual control, while a laryngoscope should be used to pass a catheter into the larynx and the trachea (Fig. 45). Sputum is aspirated from the

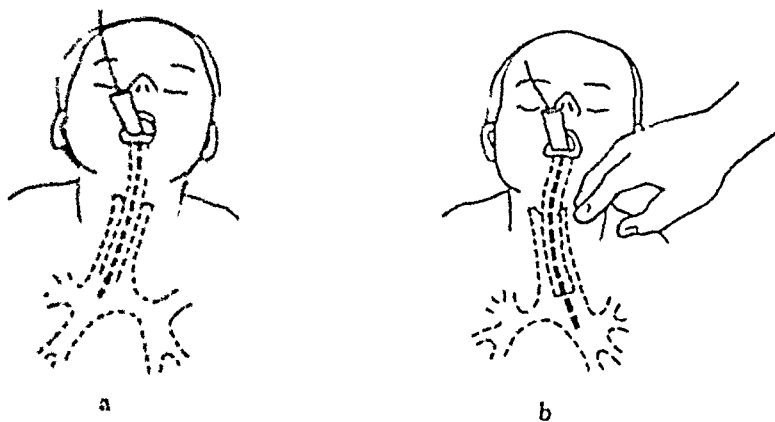


Fig 45 Intubation of the trachea and bronchi

*a*—normally the tube passes the trachea and the right main bronchus, *b*—if the trachea is displaced to the right the tube enters the left bronchus

airways using a catheter which is attached to a source of vacuum via a T-piece. The third end of the latter is closed only during aspiration of fluids, otherwise the distal end of the catheter can suck the mucosa into the tube and injure it. Any electrical or jet-ejection pump can be used to create vacuum of about 50 cm H<sub>2</sub>O. Before carrying out aspiration, it is desirable to give pure oxygen to breathe for a few minutes to prevent severe hypoxia. Aspiration should not continue for more than 10-15 seconds.

Direct laryngoscopy with catheterization of the trachea and bronchi should be carried out with halothane anaesthesia. The catheter is passed into the left bronchus by moving the trachea to the right, and on the contrary, by displacing the trachea to the left, the catheter can be passed into the right bronchus. Aspiration of fluids from the tracheobronchial tree should be conducted in aseptic conditions. Catheters and solutions used for washing the trachea and bronchi must be sterile. The size of the catheter depends on the child's age. It is important that the diameter should not be larger than  $\frac{1}{4}$  of the diameter of the main bronchus. The distal end of the catheter must be smooth and rounded, with several openings on the lateral surfaces. As a rule, the catheter is passed as far as it goes and then pulled back slightly, afterwards aspiration is started.

*Bronchoscopy* is the most effective method of cleaning the tracheo-bronchial tree. Bronchoscopy in children should be conducted with anaesthesia using depolarizing muscle relaxants. The lungs should be ventilated with pure oxygen. After complete relaxation of the muscles and hyperventilation, the bronchoscope is passed through the vocal cords (Fig 46). Further ventilation of the lungs is effected through the bronchoscopic tube. The size of the bronchoscopic tube depends on the age of the child. A smaller tube should be used for



Fig. 46 Bronchoscopy

premature infants. After the bronchoscope has passed into the bronchus or trachea, sputum is aspirated through a plastic or rubber catheter. If necessary, the trachea and bronchi are washed with solutions of sodium bicarbonate, antibiotics, antiseptics, or mucolytics, with subsequent removal of excess fluids by aspiration. Percussion massage of the corresponding parts of the chest is often carried out during aspiration. Extubation should be done on termination of action of the muscle relaxants and re-establishment of spontaneous respiration.

It should be remembered that bronchoscopy can intensify hypoxia and this operation should therefore be as short as possible. Rules of asepsis must be observed during bronchoscopy because the respiratory mucosa can be likened to an open wound as regards its susceptibility to infection.

Bronchoscopy is also used in cases where it is necessary to perform a temporary occlusion of the bronchi in pneumothorax. The bronchus is blocked using a porolon (polyurethane foam) or collagen seal, thus eliminating the air leakage through the fistula. Collagen seals will later resolve, while porolon seals must be removed during bronchoscopy.

*Prolonged nasotracheal intubation* is the common method to maintain free patency of the airways for a long period of time. Silicone-coated tubes can remain in place for weeks and even months. Tubes without cuffs are usually used for prolonged nasotracheal intubation. Intubation should be performed with anaesthesia. The tube is first passed through a nasal passage without visual control and then, using a laryngoscope and Magill forceps, the tube is directed into the trachea. If respiration is uniform over the entire surface of the lungs, the tube is fixed. Prolonged nasotracheal intubation can be successful only on condition of strict observation of asepsis rules, adequate conditioning of the breathing mixture, and permanent control of the airways patency. The tube or all objects that may contact it (catheters for aspiration of fluids, connectors, hoses, etc.) must be sterile. The breathing gas should be passed through a bacterial filter. The presence of the tube interferes with realization of the coughing reflex. Therefore, placing the child in position promoting drainage of fluids, percussion massage, exercises, and physiotherapeutic procedures become especially important. Sputum should be removed from the tube by aspiration only in case of necessity.

During normal breathing air is humidified and warmed up in the upper airways. The breathing gas should therefore be conditioned before supplying it to a child with prolonged nasotracheal intubation. Special conditioners supplied together with the apparatus for artificial ventilation of the lungs should be used for the purpose. Periodical inhalation and instillation of a few drops of isotonic sodium chloride solution into the endotracheal tube are ineffective. It has been calculated that if the breathing gas is insufficiently moist, the liquid loss with perspiration in an infant under 1 year of age can be about 1 litre (to 3 litres in older infants). These liquid losses should be considered when calculating the volumes of fluids for infusion therapy.

It should be remembered that the respiratory tube may at any time be obstructed at the place of bend, or the tube may slip out of the trachea or into the bronchus. The respiratory and cardiac functions should therefore be monitored (in addition to permanent observation by the physician or a nurse). Gas-analysing monitors should preferably be used, because impedance sensors can mistake convulsive contractions of the chest muscles in obstruction of the airways for the respiratory movements.

**Aerosol therapy.** Aerosols are mainly used for dilution and administration of medicinal preparations (mostly local) intended for inhalation. Mucolytics (substances thinning sputum) and substances with broncholytic properties, and also anti-inflammatory and antibacterial preparations are commonly given as aerosols. Aerosol therapy is indicated for acute and chronic diseases of the bronchi and lungs that are attended with accumulation of thick purulent spu-



tum and also for bronchial asthma. Aerosol inhalations are usually carried out with pneumatic or ultrasonic atomizers.

Mucolytics improve rheological properties of sputum and facilitate its evacuation. Mucolytics are commonly classified as detergents and enzyme preparations. Sodium bicarbonate is a commonly used detergent. Popular enzyme preparations are deoxyribonuclease and synthetic preparations on the basis of trypsin (chymosin and chymotrypsin). When prescribing enzymes it is necessary to remember that apart from their mucolytic effect, they have an adverse effect on the function of ciliary epithelium, they destroy the surfactant and can cause allergic reactions.

Broncholytics used for aerosol therapy are substances with adrenergic action (adrenaline, ephedrine, isadrin) and purine derivatives (euphylline). The main disadvantage of inhalation of these preparations is uneven distribution in the lungs: the larger portion of the preparations deposits in regions where ventilation is better and obstruction is smaller. Moreover, general adrenergic reactions are possible because of the resorptive effect.

Antibiotic aerosols should be administered only after testing the microflora for sensitivity to them.

Ultrasonic inhalation should be used carefully with infants because of the danger of hyperhydration. It has been shown that inhalators can provide conditions for infection spreading.

**Oxygen therapy.** Arterial hypoxaemia is the most common respiratory insufficiency. Oxygen inhalation is therefore practically an obligatory component of respiratory therapy. Like any other medicine, oxygen should be administered in the appropriate doses. A correct determination of the dose is only possible with control of fractional concentration of oxygen in the breathing gas and partial pressure in the arterial blood ( $\text{PaO}_2$ ). Depending on the condition of the gas exchange function of the lungs, the optimum oxygen dose can vary greatly for one and the same child. For example, inhalation of pure oxygen by a neonate with the disease of the hyaline membranes during the first day of life ensures  $\text{PaO}_2$  at 50-70 mm Hg. In 2-3 days the same oxygen concentration raises  $\text{PaO}_2$  to the toxic level (200-300 mm Hg). It is therefore necessary to control permanently oxygen concentration in the breathing gas and the partial pressure in the arterial blood during administration of oxygen by any method.  $\text{PaO}_2$  below 80 mm Hg in infants and 60 mm Hg in neonates indicates hypoxia, while  $\text{PaO}_2$  over 100 mm Hg indicates hyperoxygenation for all ages. Unless these data are available, the physician may not be sure that he does not do harm to the patient.

Taking blood specimens for the determination of the acid-base balance and the gas composition was discussed earlier. It is only necessary to add here that it seems reasonable to use skin sensors with neonates and infants for the purpose. The sensors can control trans-

cutaneous oxygen tension which well correlates with  $PO_2$  in the arterial blood

Nasal tubes, face masks, tents, and couveuses are commonly used for oxygen inhalation in the paediatric clinic

*Nasal tubes* can be twin or single, the former being intended for passage into both nostrils. Single nasal tubes have numerous openings at their distal ends and are often introduced nasopharyngeally. Nasal tubes have the following disadvantages (1) it is impossible to attain high oxygen concentration with them, (2) nasal breathing is difficult and the tubes irritate the respiratory mucosa, (3) concentration of oxygen in the breathing gas cannot be controlled

Various oxygen concentrations (high concentration included) can be attained with *face masks*. Special masks with calibrated openings for gas exchange with the atmosphere are available. It is convenient to adjust the required oxygen concentration by passing the gas mixture at specified rates as indicated on the face mask. The disadvantage of such masks is their difficult fixation on the face and the possibility of leakage

*Oxygen tents* have remained the most common means of administering oxygen. The child feels comfortable in a tent and the concentration of oxygen in the breathing gas can easily be maintained at the required level. But since an oxygen tent cannot be sealed (separated from the environing atmosphere) the oxygen delivery rate should be rather high (8-12 l/min), but even at this rate it is difficult to raise oxygen concentration to 50-60 per cent

Oxygen therapy is administered to neonates straight in a *couveuse*. Closed couveuses can be used to raise oxygen concentration to the wanted level. But opening couveuses even for a short time quickly results in the fall of oxygen concentration

When conducting *oxygen therapy*, especially in infants, it is necessary to control the temperature and humidity of the breathing gas. It has been established that if the oxygen concentration exceeds 40 per cent, the breathing gas should be humidified to prevent liquid loss with perspiration. Infants are very sensitive to cold and if cold air washes the infant's face, he reacts as if the entire body is cooled. It is therefore necessary that oxygen is passed through a preheater (a container filled with hot water) to warm the gas before it enters the infant's airways

Retrolental fibroplasia (retinopathy of premature infants) and bronchopulmonary dysplasia are the most severe complications of oxygen therapy in children. Retrolental fibroplasia develops in the presence of hyperoxaemia in premature infants. High  $PaO_2$  causes persistent contraction of the retinal vessels. In 1-2 months after termination of oxygen therapy the retinal vessels dilate and proliferate, oedema of the retina develops. In some cases the disease is cured without leaving any organic changes, but in about 25 per

cent of neonates, retinal detachment occurs with subsequent cicatrization and blindness. The only clinical sign of the early stage of this disease is contraction of the retinal vessels. Therefore, prevention of this severe complication includes maintaining the appropriate  $\text{PaO}_2$  level.

Bronchopulmonary dysplasia is the result of toxic action of oxygen on the pulmonary tissue. As distinct from retrolental fibroplasia, the aetiological factor of dysplasia is not hyperoxaemia but the high oxygen concentration in the breathing gas. Histologically the lung affection is characterized by oedema and proliferation of cells of the alveolar membrane with subsequent fibrosis. The length and intensity of oxygen therapy causing this disease have not been established. Most researchers believe that oxygen concentrations below 50 per cent are safe. Higher concentrations increase the risk of development of bronchopulmonary dysplasia in proportion to the oxygen concentration and the time during which oxygen therapy is administered.

Death rate of this disease is 30-50 per cent. Oxygen must therefore be administered in the minimum possible doses and during the shortest possible time.

**Helium-oxygen therapy.** The low density of helium accounts for its use in oxygen-helium therapy. The air passes the airways of man in laminar currents. But if an air stream encounters an obstacle (oedema of the subglottic space, accumulation of sputum), the gas flow becomes turbulent. The aerodynamic resistance and the work of respiration increase considerably. If helium is substituted for nitrogen, the density of the gas mixture decreases and the vortex resistance diminishes. Helium-oxygen mixtures are therefore used to decrease the work of respiration in children with stenosed larynx, trachea, or large bronchi. Attempts to use helium for treatment of other respiratory disorders, such as bronchial asthma, asphyxia of neonates or diffuse microatelectasis, were a failure.

Helium-oxygen inhalation is simple. The gases are delivered into a tent from cylinders through rotametric flowmeters. Numerous studies have shown that the most effective helium to oxygen ratio is 3:1. Inhalations are usually conducted 2-5 times a day in 1-2-hour sessions.

The disadvantage of helium-oxygen therapy is the danger of hypothermia, because the specific heat of helium is several times higher than that of air. Moreover, substitution of helium for nitrogen can in some cases accelerate the alveolar collapse and cause microatelectasis.

**Spontaneous respiration under constant positive airway pressure (CPAP).** This is a comparatively new method of treating arterial hypoxaemia. This method was introduced in 1971 by Gregory after his intensive research, which showed that moderate distension of

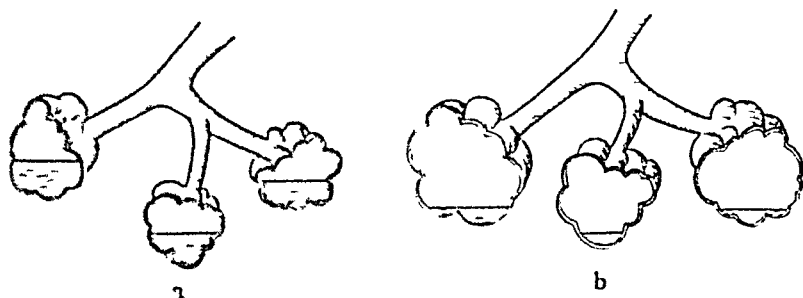


Fig 47 Spontaneous respiration with constant positive airway pressure  
*a*—alveoli during normal respiration, *b*—alveoli during spontaneous respiration with CPAP

the lungs improves radically the results of treatment of neonates with the disease of hyaline membranes. It was established later that constant positive airway pressure is effective in treating any disease causing hypoxaemia and decreasing lung elasticity (oedema of the lungs, aspiration syndrome, condition after thoracotomy, and the like). CPAP is also used to prevent and correct post-operative respiratory disturbances in children, and also to decrease the toxic effect of hyperbaric oxygenation.

*Physiological substantiation of CPAP.* Positive pressure prevents both expiratory closure of the airways and the alveolar collapse, the latter by compensating for the decreased surfactant level. The functional residual volume of the lungs increases at the expense of stretching hypoventilated and collapsed alveoli. The ventilation-perfusion ratio normalizes and the intrapulmonary venous-arterial shunting decreases (Fig 47).

Improved diffusion capacity of the lungs during CPAP is associated with decreased oedema of the alveolar-capillary membrane. The alveolar-arterial gradient of oxygen falls. All these changes cause a marked increase in partial tension of oxygen in arterial blood with unchanged oxygen concentration in the breathing gas.  $\text{PaCO}_2$  often decreases as well, especially in the presence of hypercapnia. It should be remembered that inflation of the lungs always increases the anatomical dead space. Therefore, even unchanged  $\text{PaCO}_2$  indicates improvement of alveolar ventilation.

The study of respiratory mechanism has shown that during CPAP the dynamic distensibility of the lungs decreases by 20-30 per cent of the initial level. This can be explained by the fact that during distension of the lungs not only the hypoventilated and atelectasis-affected alveoli are distended, but normally functioning alveoli become overdistended too. The aerodynamic resistance in children with lung diseases decreases  $1\frac{1}{2}$ -2 times during CPAP. Positive pressure in the airways does not practically influence the venous

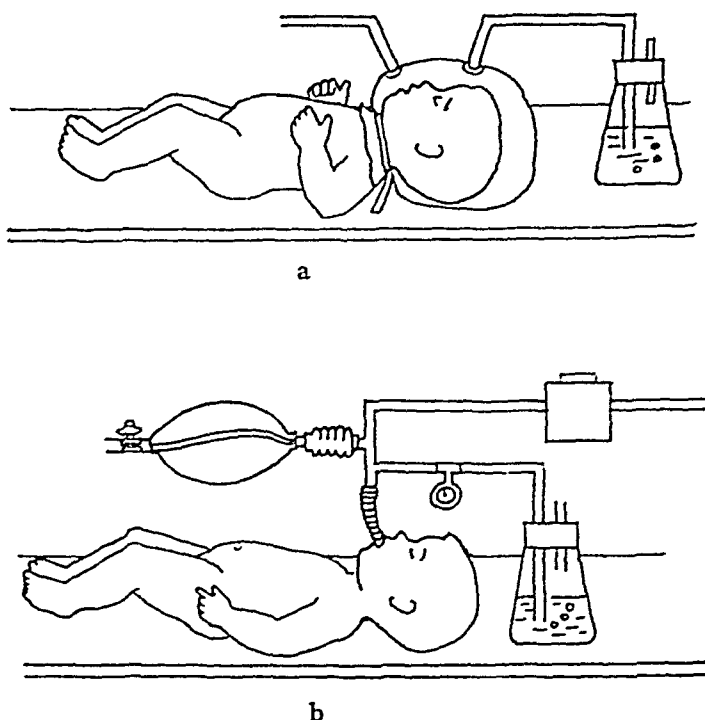
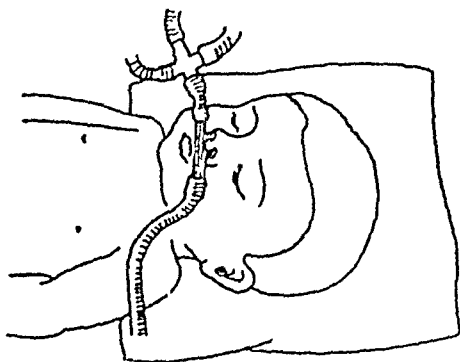


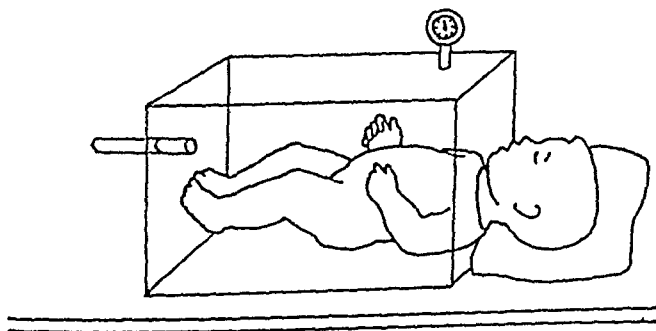
Fig 48 Spontaneous respiration with constant positive airway pressure  
*a*—CPAP with a plastic bag, *b*—Gregory's method, *c*—respiration through nasal tubes, *d*—continuous negative chest wall pressure

backflow in children with normal circulating blood volume. Quite the contrary, the cardiac output increases in most cases, which can be explained by elimination of hypoxia and improved contractility of the myocardium.

**CPAP techniques** Positive pressure in the airways can be attained by various methods (Fig 48). The method proposed by Gregory is as follows: a cross-piece is connected to the endotracheal tube and a rubber bag. The bag has an outlet, which is closed by a Moor clamp. Two tubes are connected to the cross-piece: one tube supplies the air-oxygen mixture, while the other leads to a pressure gauge and a water seal. The breathing gas can be supplied from rotametric flowmeters; the oxygen concentration can be adjusted between 21 and 100 per cent. In order to prevent accumulation of carbon dioxide in the system, the gas should be supplied at a rate 3-4 times exceeding the minute ventilation volume of a child (4-6 l/min for infants under 1 and 8-12 l/min for older children). Using the Moor clamp, the gas flow from the bag is so adjusted that the required positive pressure (as read off the pressure gauge) is maintained in the system. If the pressure exceeds the preset value, excess gas is released through the water seal into the atmosphere. The breathing gas should be



c



d

humidified and warmed up because it is delivered into the lungs through the endotracheal tube without being warmed in the upper airways. Humidifiers of artificial respiration apparatuses can be used for the purpose.

The Gregory method is commonly used with critical patients and CPAP is maintained for prolonged periods of time. This technique is suitable when the patient is no longer assisted by a respirator and also for correcting respiratory disorders in the early post-operative period.

Techniques are known by which CPAP is delivered to nonintubated patients. By one of them, a plastic bag is put on the child's head and positive pressure is created in it. The size of the bag depends on the size of the child's head; when the bag is inflated, its walls should be at a distance of 5-10 cm from the patient's face. This ensures adequate ventilation of all parts of the bag. Two tubes lead to the bag: one of them is connected to the source of breathing gas, while the other to the water seal. It is desirable that the diameter of the gas delivery tube should be not less than 1 cm to prevent excessive

noise inside the bag that may annoy the child. A Dobro filter can be used as a water seal. The height of the air column in the tube indicates the amount of positive pressure in the respirator circuit. The bag should be sealed at the child's neck using a wide porcelain tape (several turns round the neck). The pressure of the band on the neck should be only slight in order to prevent disturbance in the blood circulation or oedema of the head tissue. Excess gas is released from the bag mostly in the region of the child's neck and partly through the hydraulic seal. The disadvantage of this method is possible aerophagia and overdistension of the stomach. To prevent this complication a gastric tube should be passed into the stomach and its distal end left open.

Face and nasal masks can be used to create positive pressure in the airways. The mask is connected with a to-and-fro apparatus for anaesthesia and is held to the face of neonates by an elastic net (or by rubber bands to the face of older children). But it is often difficult to ensure a tight seal between the mask and the face, while excessive pressure of the mask on the face quickly causes maceration of epithelium.

Nasal prongs can be used to deliver CPAP to neonates, who usually breathe through the nose and keep their mouths closed until the pressure in the airways exceeds 10-12 cm  $H_2O$ . The disadvantage of the method is hypersecretion and injury of the nasal mucosa if the prongs remain in the nostrils for a long time.

Special chambers are used to create constant negative pressure over the child's chest. The pressure inside the child's respiratory system will then be higher than inside the chamber. The child can breathe either ordinary air or an air-oxygen mixture. Such chambers are usually placed in a couveuse where a special climate is provided. The disadvantage of this method is the difficult access to the child and the risk of cooling the body by a permanent air movement in an untight chamber (Fig. 49).

*Indications for CPAP and its techniques* The indications for CPAP are severe arterial hypoxaemia ( $PaO_2$  below 60 mm Hg) during breathing 50 per cent air-oxygen mixture connected with disturbed ventilation-perfusion ratio, high intrapulmonary shunting, and decreased distensibility of the lungs. These disturbances usually occur in the disease of hyaline membranes, lung oedema, aspiration syndrome, and after injurious surgery on thoracic and abdominal organs. Moreover, CPAP is helpful during transition from artificial to spontaneous respiration, and also during hyperbaric oxygenation to lessen the toxic effect of oxygen on the lungs.

CPAP usually begins with inhalation of mixtures containing from 80 to 100 per cent oxygen under a pressure inside the airways of 3-5 cm  $H_2O$ . Later, as the intrapulmonary gas exchange improves, the oxygen concentration in the mixture is gradually decreased.

(with  $\text{PaO}_2$  control) to a non-toxic concentration (50 per cent) The pressure in the bag is then decreased in 1-2 cm  $\text{H}_2\text{O}$  steps If  $\text{PaO}_2$  falls below 60 mm Hg, the pressure is raised to the previous level The procedure should be discontinued when  $\text{PaO}_2$  stabilizes above 60 mm Hg, the oxygen concentration in the breathing gas being less than 50 per cent and the pressure inside the system being atmospheric The patient should then be placed in an oxygen tent

*Complications* Complications of CPAP occur in 2-3 per cent of cases Increased pressure inside the respiratory system may cause pneumothorax, pneumomediastinum and pulmonary interstitial emphysema According to the data available, the incidence of pneumothorax in neonates with the disease of the hyaline membranes (after delivered CPAP) is 2 times higher than with ordinary respiration CPAP may cause a marked reduction of the cardiac output and hypotension in children with severe circulatory disorders (especially in the presence of dehydration and hypovolaemia with the deficit of the circulating blood volume exceeding 30 per cent)

*Contraindications* CPAP is contraindicated for bronchopleural fistulae, intense pneumothorax and marked hypoventilation ( $\text{PaCO}_2$  above 60 mm Hg, with a tendency to increase) Inflation of the lungs is ineffective in pronounced purulent tracheobronchitis and non-homogeneous affections of the lung tissues, in most forms of pneumonia included

*Artificial lung ventilation* This is a temporary substitution for the external respiratory function This technique is now practiced in many hospitals and maternity houses Timely and correct artificial lung ventilation in severe respiratory disorders in children is decisive for good outcomes of the disease But artificial ventilation of the lungs in neonates and infants is a complicated problem, which is first of all connected with their special anatomical and physiological properties Small respiratory volumes, the high gas flow-rates with high resistance of the airways require not only special apparatuses but also special training

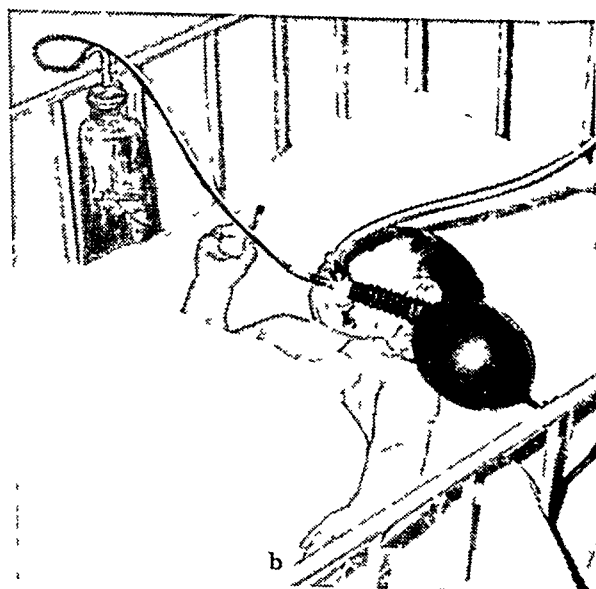
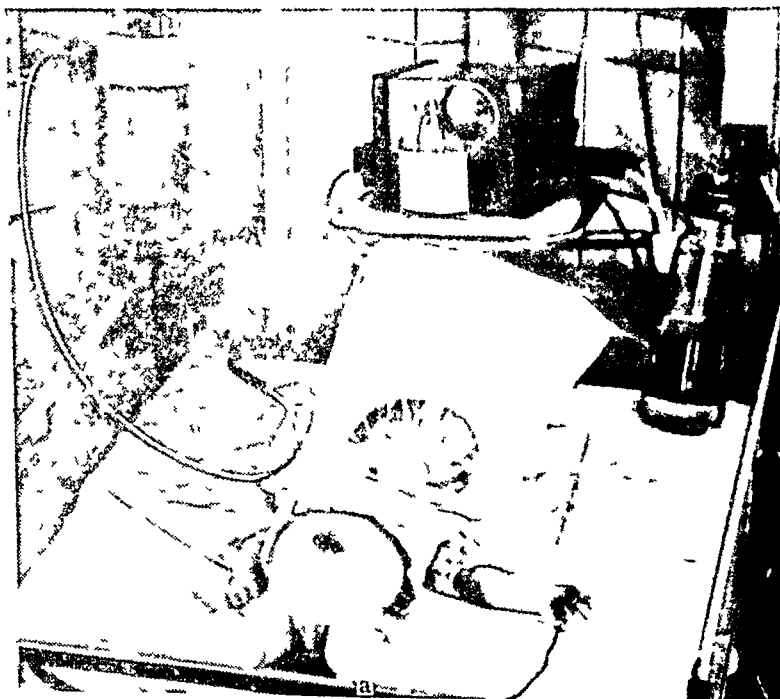
*Causes of respiratory dysfunction* Various respiratory disorders are often the main cause of neonatal mortality Timely artificial ventilation of the lungs is therefore an important problem

Acute respiratory insufficiency in infants can be caused by the following

I Pathology of the lungs

- a) hyaline membrane disease, diffuse atelectasis, oedematous-haemorrhagic syndrome,
- b) pneumonia of various aetiology (intrauterine, aspiration, viral-bacterial, staphylococcal),
- c) congenital defects of the lungs (hypoplasia or agenesis, lobar emphysema),





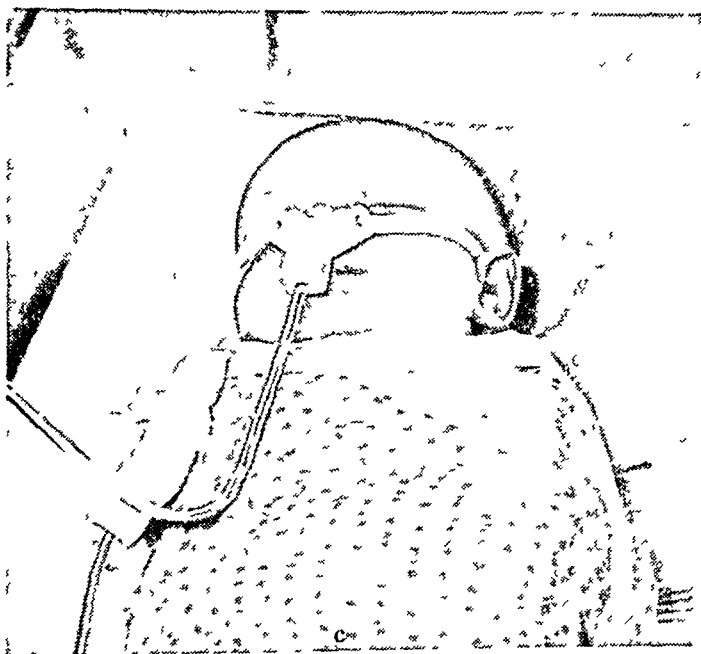


Fig 49 Conducting spontaneous respiration with constant positive airway pressure

*a*—CPAP with a plastic bag, *b*—Gregory's method, *c*—through intranasal cannulas

d) congestion in the lungs in the presence of congenital heart defects (aortal coarctation, great vessel transposition, open ductus arteriosus, great defect of the interventricular septum), in the left ventricular heart failure and primary myocarditis

## II Pathology of the airways

a) obstruction of the airways by aspired amniotic fluid or gastric contents, the condition mostly occurs in neonates after severe hypoxia, with affection of the central nervous system and congenital defects of the gastrointestinal tract, aspiration of foreign bodies,

b) obstruction of the airways with sputum during infections, bronchospasm and oedema of the mucosa,

c) congenital defects of the airways (atresia of the choanae, the Pierre-Robin syndrome, cleft hard and soft palate)

## III Extrapulmonary causes of acute respiratory insufficiency

a) affections of the central nervous system due to intracranial birth trauma, intoxication, disordered haemo- and liquor dynamics, residual effects of anaesthetics and narcotic analgesics,

b) myopathies (myasthenia, myotonic dystrophy, etc ),

c) restrictive disturbances of respiration (pneumo- and haemothorax, diaphragmatic hernia, high diaphragm),

d) immaturity of the central regulation of respiration in premature neonates

*Pathophysiological effects of artificial lung ventilation* As a method of intensive respiratory therapy, artificial lung ventilation has a complicated effect on various organs and systems of the body. Successful artificial ventilation of the lungs is possible only with adequate knowledge of the processes occurring inside the body. Effective artificial lung ventilation in children with respiratory insufficiency eliminates hypercapnia and hypoxia, which is connected with increasing ventilation volume, improved ventilation-perfusion ratio and decreased intrapulmonary shunting. Elimination of respiratory acidosis and gradual normalization of pH promote restoration of exchange processes. Catecholæmia disappears, microcirculation improves, the blood of the depots is involved in the general circulation. Elimination of hypoxia and improvement of myocardial contractility also promote normalization of blood circulation.

But apart from the first positive effects of artificial pulmonary ventilation, undesired pathological changes also occur. Lung ventilation with forced insufflation of air into the lungs changes substantially the mechanism of respiration. As distinct from spontaneous lung ventilation, intra-alveolar and intra-pleural pressure becomes positive at the end of inspiration in artificial lung ventilation, the dynamics of the transpulmonary pressure changes accordingly. Changed conditions of ventilation cause some pathophysiological shifts, which result in decreased distensibility of the lungs and increased aerodynamic resistance of the airways. The disordered mechanism of coughing and decreased function of ciliary epithelium during artificial ventilation inevitably impair withdrawal of sputum. This, in turn, increases the risk of microatelectases if the activity of the surfactant is low. These, together with the changes in regional ventilation-perfusion ratio in the lungs caused by prolonged motionless position of the patient, can cause severe disorders in the intrapulmonary gas exchange.

The absence of the drawing effect of the chest during artificial pulmonary ventilation can interfere with the venous outflow and decrease the cardiac output, but this usually occurs in patients with markedly decreased volume of circulating blood. Moreover, increased airway pressure disturbs the pulmonary blood circulation, circulation of the lymph, and in some cases promotes the development of oedema and transudation of fluids inside the alveoli. Therefore, in order to ensure successful artificial ventilation of the lungs for a long period of time, the physician should be well aware of all possible pathophysiological shifts that can occur in the body during mechanical ventilation. Using optimum conditions and observation of all necessary conditions minimize the harmful effects of artificial pulmonary ventilation and ensure successful treatment.

*Indications for artificial pulmonary ventilation* An absolute indication for artificial pulmonary ventilation in children is the absence of spontaneous respiration (apnoea) irrespective of the causative factor. In these conditions artificial ventilation of the lungs is a component of the resuscitation measures. Another indication for artificial lung ventilation is a pathological arrhythmia of respiration, which usually occurs in affections of the central nervous system, in oedema and injury of the brain, severe intoxications and infections.

The assessment of indications for artificial lung ventilation becomes complicated in conditions of progressive symptoms of hypoventilation. The clinical signs that can be considered as indications for artificial lung ventilation in children are increasing cyanosis, acceleration of respiration (two times exceeding the normal rate), and involvement of the accessory muscles in the respiratory act. The most informative criteria which indicate the necessity of artificial lung ventilation are the acid-base balance and blood gas findings.

1 Hypercapnia  $\text{PaCO}_2$  is above 60 mm Hg or increases progressively by more than 10 mm Hg per hour.

2 Hypoxaemia  $\text{PaO}_2$  is below 50 mm Hg in neonates and below 70 mm Hg in older children (the oxygen to air ratio in the breathing gas being 1 : 1).

3 Acidosis The pH is below 7.2, despite the administration of sodium bicarbonate in a dose of 2-4 mmol per kg of body weight.

*Techniques of artificial lung ventilation* Blowing breathing gas into the patient's lungs is the most common method of giving artificial respiration. This can be done by the mouth-to-mouth technique, or using an air bag, or an apparatus for giving anaesthesia, or special apparatus intended for artificial lung ventilation. The choice depends on many circumstances, such as availability of apparatus, time during which the artificial ventilation should be conducted, and the skill of the medical personnel. The mouth-to-mouth method is used in emergency cases, in the absence of special tools and apparatuses. It should be continued until spontaneous respiration is restored or other means of giving artificial respiration become available.

Artificial ventilation of the lungs is easier with hand respirators, self-expanding bags, bellows, etc. An air bag has a system of valves, by which the breathing gas is pumped into the lungs and the exhaust air is discharged into the atmosphere. A special connector is used through which oxygen can be delivered into the bag together with air.

When giving artificial ventilation, either by the mouth-to-mouth technique or by a respirator, the patient's airways should be cleaned to ensure their patency.

Artificial ventilation can be given by an oro-nasal mask, airways

or by an endotracheal tube. Some air bags can be used to create positive end expiratory pressure in the lungs, which is very important for neonates with rigid lungs. Artificial ventilation by a bag or with manual interruption of the compressed gas flow by T-piece (Ayre method) is usually conducted during operative anaesthesia.

*Automatic ventilation of the lungs* Various apparatuses are used for artificial ventilation of the lungs in children. Their operation can be controlled by volume, pressure or time. The choice often depends on personal experience of the physician and availability of particular instruments.

*Conditions of artificial lung ventilation* The following three techniques are commonly used with children: 1) intermittent positive pressure ventilation, 2) intermittent positive pressure ventilation with positive end expiratory pressure, 3) intermittent mandatory ventilation, during which the patient is given only several forced inspirations, while he breathes spontaneously between these periods. This type of ventilation is only possible with ventilators in which gas is circulated at a constant rate. The patient can thus be 'taught' to breathe independently of the ventilator before he is allowed to breathe spontaneously.

*General principles of artificial lung ventilation* The child is connected to the ventilator through an endotracheal tube or a tracheostomic cannula. Thermoplastic tubes without inflatable cuffs are commonly used for nasotracheal intubation. The tubes should be changed once a day or every other day, depending on the amount and character of sputum. Sterile tubes need no anti-inflammatory or anaesthetic coating. The intubated child should be auscultated thoroughly to be sure that respiration is adequately conducted over the entire lung surface. The tube should then be fixed and its position checked again by auscultation or x-raying.

Ventilation conditions can be determined using Redford or Engstrom-Herzog-Norlander nomograms. But practice shows that pre-calculated figures almost never ensure adequate ventilation of lungs in a particular patient. The ventilation conditions and the oxygen concentration in the breathing gas can finally be obtained only after determining the blood gas status and the acid-base balance. The common practice is to connect the child to a ventilator, to establish the respiratory rate corresponding to the child's age, and adjust the tidal volume at the level at which the maximum inhalation pressure should be 20-25 cm H<sub>2</sub>O. The oxygen concentration in the breathing gas should be adjusted from 50 to 100 per cent depending on the degree of cyanosis. The acid-base balance and gas composition of the blood should be analysed in 10-15 minutes. In case of hyperventilation (PaCO<sub>2</sub> below 30 mm Hg) the inspiratory pressure ( $P_{i,n}$ ) should be decreased by 3-5 cm H<sub>2</sub>O, frequency of respiration ( $f$ ) should be decreased accordingly. In hypoventilation  $f$  and  $P_{i,n}$

should be increased accordingly. If blood analysis shows that hypoxaemia persists in a child ( $\text{PaO}_2$  below 60 mm Hg), positive end expiratory pressure (5-7 cm  $\text{H}_2\text{O}$ ) should be provided, and the inspiration to expiration ratio increased to 1 : 1 or 2 : 1. The concentration of oxygen in the breathing gas should also be increased if possible. Further on, as the condition of the child improves, the following should be ensured: the oxygen concentration in the gas should be rapidly decreased to a non-toxic level (below 50 per cent), the end expiratory pressure should be gradually decreased to zero and the inspiration to expiration ratio adjusted to 1 : 2. Only then the respiration frequency can be decreased and the child prepared for weaning from ventilatory support. These are the main principles that a physician should observe in selecting conditions for pulmonary ventilation.

It is often difficult to synchronize the child's respiration with the work of the ventilator. A simple and effective method is periodical manual ventilation of the lungs using an airbag. The procedure should be conducted for 5-10 minutes before starting mechanical ventilation, before and after aspiration of sputum from the airways, and also in cases of asynchronous respiration. Adaptation of the child to the ventilator can be ensured by maintaining moderate hyperventilation. This method should not however be abused because hyperventilation can cause pathophysiological reactions in a child. If the absence of synchronism impairs the child's condition and  $\text{PaCO}_2$  increases, sedatives and muscle relaxants (sodium oxybate in a dose of 100 mg/kg, diazepam in a dose of 1 mg/kg, and tubocurarine in a dose of 0.3-0.4 mg/kg) can be administered.

*Observation and care of patients with artificial lung ventilation*  
A child requires permanent care and observation during artificial ventilation of his lungs. Any disturbance in the apparatus, leakage or obstruction of the endotracheal tube can be lethal. Therefore, in addition to permanent visual observation of the child's general condition, it is necessary to monitor his heart rate, respiratory frequency, tidal volume, oxygen concentration in the breathing gas, its temperature and humidity. Since some humidifiers do not meet the exacting requirements, it is recommended to introduce 0.5-1 ml of sterile isotonic sodium chloride solution into the endotracheal tube (once an hour, dropwise).

Mucus should obligatorily be removed from the trachea and bronchi by aspiration. This manipulation should be performed in aseptic conditions. The patient should be disconnected from the apparatus and 1-2 ml of isotonic sodium chloride solution introduced into the tube. Then several inspirations should be conducted using an air bag, and a sterile catheter should be passed into the endotracheal tube with rotation. Aspiration of mucus should not last longer than 8-10 seconds. During this procedure an assistant should give massage

and knock slightly on the child's chest. Then ventilation with a hand air bag should again be conducted for several minutes and the child can then be connected to the apparatus.

To prevent atelectasis and pneumonia, the child should periodically be turned from one side to another, placed in positions improving drainage of fluids, his chest should be massaged and physiotherapy given. These measures should be conducted regularly, at intervals not exceeding 3-4 hours.

The acid-base balance and gas status of the blood should be determined each time after changing the ventilation conditions. If these conditions are stable, the blood analyses should be conducted not less than 3-4 times a day. When determining the ventilation conditions for children with severe respiratory insufficiency, blood specimens should be taken from the artery (radial, temporal, umbilical, or femoral). Arterialized capillary blood may only be taken for follow-up observations. The problem can be facilitated by determining transcutaneous  $PO_2$ , which well correlates with findings of the arterial blood.

During artificial lung ventilation special attention should be paid to infusion therapy. Amounts of infused electrolytes and other fluids should comply with the physiological demands and liquid losses. Solutions of glucose, amino acids, fat emulsions, albumin, plasma, and electrolytes are used for infusion therapy. Nutrition of patients with artificial ventilation of the lungs should preferably be parenteral, but if the child's condition improves gradually, food can be administered through a gastric tube into the stomach or duodenum.

Artificial ventilation of the lungs in children can only be successful with strict observation of asepsis and antisepsis regulations at all stages. Any object that may contact the respiratory organs of the child should be sterile: endotracheal tubes, connectors, humidifiers, catheters for aspiration of mucus, etc. Parts of modern apparatus for artificial lung ventilation can be treated in an autoclave. In order to prevent gram-negative infection, it is recommended to change daily the airways and the humidifier. Bacterial air filters have recently been introduced into practice. Prophylactic use of antibiotics in children with artificial ventilation of the lungs is argued and their efficacy depends on particular conditions.

Weaning from ventilatory support should be a planned procedure and conducted only during the day time. The design of the ventilator permitting, the child should first be allowed to breathe with intermittent mandatory ventilation of his lungs with gradually decreasing respiration rate to 10-5 per minute. The child is then allowed to breathe spontaneously with constant positive pressure of about 3-5 cm  $H_2O$ , the oxygen concentration in the gas should be 10 per cent exceed its concentration in the gas used during artificial venti-

lation of the lungs. If there are no signs of developing respiratory insufficiency during the course of at least 12 hours and the gas composition of the blood remains normal, the child can be extubated.

*Dangers and complications of artificial lung ventilation* Some negative effects of artificial lung ventilation have already been described. It should be noted that the inhibiting effect of this procedure on blood circulation of neonates is less pronounced than in older children, cases of cardiovascular depression are rare. At the same time neonates are much more sensitive to high oxygen concentrations and increased pressure in the respiratory system.

Frequent complications of artificial lung ventilation are pneumothorax and pneumomediastinum. These complications are more frequent with positive end expiratory pressure in neonates borne underweight. Diagnosis of pneumothorax is not difficult provided the condition of the respiratory system of the child is permanently controlled. In cases with small pneumothorax (without symptoms of intrathoracic tension), or in cases with pneumomediastinum, it is sometimes sufficient only to observe the child carefully. But if the amount of air in the pleural cavity is large, or in cases with valvular pneumothorax, it is necessary to puncture the pleural cavity immediately.

*Hyperbaric oxygenation* This method is based on the medicating effect of super-high partial pressure of oxygen, which promotes dissolution of excess amounts of oxygen in blood plasma. Hyperbaric oxygenation is widely used today for intensive therapy of conditions associated with severe hypoxia. The medical effect of hyperbaric oxygenation for elimination of various forms of hypoxia is first of all explained by purely physical laws of solubility of gases in liquids.

Under normal atmospheric pressure haemoglobin of blood is practically completely saturated with oxygen. Normally 100 ml of arterial blood contain about 20 ml of oxygen, of which only 0.3 ml is dissolved in the plasma, while the rest oxygen is bound with haemoglobin. Inhalation of pure oxygen completes saturation of haemoglobin and then the amount of oxygen dissolved in the plasma increases. The growth of oxygen solubility in blood and tissue fluids obeys Henry's law, according to which solubility of a gas in a solution under constant temperature is proportional to the partial pressure of the gas. The solubility of each gas in a mixture depends on the partial pressure of a given gas and the gases dissolve in the liquid independently of one another (Dalton-Henry law).

When applied to the human body, an increase in the partial pressure of oxygen in the alveoli involves its increasing concentration in the blood of the pulmonary capillaries and its increasing solubility in the plasma. This in turn increases considerably the



oxygen absorbability in liquid media of the body and increases the oxygen content of the cells suffering from hypoxia. According to calculations, an increase in the pressure of inhaled oxygen of 1 atm increases the oxygen solubility in the blood plasma by 0.3 ml per 100 ml (at constant temperature). It follows therefore that under a pressure of 3 atm more than 6 ml of oxygen is dissolved in 1000 ml of the blood, which corresponds approximately to the arteriovenous difference with respect to oxygen, i.e. to the normal oxygen consumption by the body at rest. The oxygen demands of most tissues will be satisfied at the expense of dissolved oxygen reserves. The role of haemoglobin as a donor of oxygen will considerably decrease in these conditions.

Hyperbaric oxygen therapy considerably increases the oxygen content of blood and of other body fluids. This ensures oxygenation of ischaemic tissues even in conditions of severe circulatory failure, haemoglobin pathologies, or other conditions under which the transport and utilization of oxygen are impaired.

*Hyperbaric oxygenation in clinical practice.* Vast experience has now been accumulated in clinical use of hyperbaric oxygenation in various branches of medicine. It is very effective in the treatment of gas anaerobic infection, severe septic and shock conditions of various genesis, and in acute exogenic intoxications.

This method can be very effective as a component of complex therapy of some critical conditions. Direct indications for hyperbaric oxygen therapy of children are as follows: 1—post-resuscitation conditions, 2—hypoxic oedema of the brain, 3—hepatic and renal failure, 4—severe combined injury, 5—severe acute exogenic intoxications, 6—air embolism; 7—long-healing wounds.

Our experience shows that hyperbaric oxygen therapy is effective in phlegmona of neonates, abscesses and purulent cephalhaematomas, diffuse purulent peritonitis, ulcerous necrotic enterocolitis, chemical burns of the oesophagus, osteomyelitis, and diffuse phlegmona.

*Risks associated with hyperbaric oxygenation.* These are mainly connected with possible toxic effects of oxygen. It is believed that the toxic effect of oxygen under high partial pressures usually manifests in the form of acute and chronic oxygen poisoning. *Acute oxygen intoxication* is caused by comparatively short exposures to oxygen under pressures higher than 3 atm. This form is characterized by affections of the central nervous system and epileptiform convulsions. Signs of intoxication disappear as the partial pressure of oxygen is decreased. If the exposure continues, irreversible changes in the central nervous system occur and the victim dies. This is a rare form of oxygen poisoning, because therapeutic doses of oxygen used in clinical practice are much lower. Some patients adapted to chronic hypoxia may develop signs of acute oxygen poisoning in conditions of normal therapeutic hyperbaric oxygenation.

*Chronic oxygen poisoning* develops after prolonged exposure to comparatively low oxygen pressures. This form of poisoning is manifested by the oxygen pneumonia. The genesis of this pneumonia is characterized by destruction of surface-active phospholipids (pulmonary surfactants) by oxygen. The pulmonary tissue collapses and atelectasis develops along with peribronchial oedema and oedema of alveolar epithelium with the loss of elasticity of the alveolar wall.

In view of possible oxygen intoxication, hyperbaric oxygen therapy is contraindicated for patients with pneumonia and epilepsy (in the anamnesis). Prolonged exposure of neonates to hyperbaric oxygen is prohibited because it can cause severe affections of the organ of vision (atrophy of the optic nerve, retrolental fibroplasia, etc.)

## Chapter 18

### Intensive Therapy of Acute Circulatory Failure

Acute circulatory failure is the condition characterized by inability of the cardiovascular system to maintain adequate blood circulation in the bodily organs and tissues.

Circulatory failure can be caused by cardiac or vascular insufficiency. These two often concur, but it is possible to establish prevalence of this or that insufficiency over the other at any given stage of the disease, this is very important for rendering necessary urgent aid.

#### ACUTE HEART FAILURE

Heart failure is the condition characterized by inability of the heart to perform the work sufficient to maintain adequate circulation of blood. Two main mechanisms are involved in the onset of heart failure, which impair the myocardial contractility. By one mechanism, the myocardium fails to contract properly due to the overload on the heart whose compensatory capacity is exhausted. This is the haemodynamic failure of the heart. It arises as a result of overload on the heart with congenital or acquired defects, in metabolic disorders in a hypertrophied myocardium, and less frequently in the presence of hypertonia of the lesser or greater circulation. The condition develops gradually and becomes chronic.

By the other mechanism the heart failure is secondary to energy and metabolic disorders in the heart muscle. This type of heart failure occurs mostly in children and is, as a rule, acute.

*Aetiology* Acute heart failure in children is usually secondary to bacterial and toxic affection of the myocardium in toxic pneumo-

nia, influenza, intestinal infections, poisoning, rheumatic myocarditis and heart defects, diphtheritic and typhoid myocarditis, acute nephritis, and long-standing anaemia

Right ventricular heart failure can develop in severe bronchial asthma, chronic pneumonia, right heart defects, lung emphysema, and spontaneous pneumothorax, it can occur during operation because of the rapid transfusion of citrate blood without simultaneous administration of calcium which prevents spasm in the lesser circulation vessels and weakening of the myocardium by neutralizing sodium citrate. Radiopaque substances, some hypertonic solutions (glucose) can cause marked contraction of the lesser circulation vessels

Vitamin B deficiency and some cases of electrolyte disbalance, especially if the potassium content is concerned, can also become the cause of heart failure. Intravenous infusion of excess amounts of blood, its plasma, salt solutions, etc., without control of the venous pressure can also become the cause of acute overload on the heart, especially in pneumonia patients

The *clinical symptoms* of acute heart failure are as follows

1—tachycardia developing as a compensatory reaction of the heart to decreased myocardial contractility and diminished stroke volume (to maintain adequate minute volume),

2—dyspnoea, which can also be a compensatory reaction. Progressive heart failure upsets the gas exchange in the lungs to intensify dyspnoea with involvement of the accessory muscles in the respiratory act,

3—enlargement of the heart; this is a very important symptom. It is very important not only to outline the heart borders but also to establish if this enlargement is the result of dilatation, compensatory or myogenic hypertrophy,

4—cyanosis of the skin and mucosa due to decreased blood supply to the tissues and their insufficient oxygenation. Metabolic processes in tissues are thus impaired to increase the proportion of anaerobic glycolysis with accumulation of products of incomplete destruction and with the shift of the reaction to acid side,

5—pasty character of the skin and oedematous tissues. Among various pathogenetic mechanisms of development of these symptoms important are blood congestion in the greater circulation, changes in the hydrostatic and colloid-osmotic pressure, increased permeability of the vascular walls, slowed blood flow in the kidneys, and electrolyte shifts caused by aldosterone hypersecretion,

6—enlargement of the liver, which indicates disturbed venous outflow, blood congestion in the greater circulation, and is attended with increased central venous pressure and development of the venous vessels on the face and chest,

7—dyspepsia

Heart failure in infants has special features. It is usually manifested by irritability of the child, difficult feeding (refusal of food), restlessness, and deranged sleep. Sometimes, the first signs of heart failure are vomiting, abdominal pain and distension. Infants usually do not develop significant oedema even in the presence of marked heart failure. Most common signs of oedema are puffy face and eyelids, swelling of the anterior fontanelle, pastiness in the region of the scrotum and sacrum. Enlargement of the heart is not obligatory.

*Diagnosis* of the heart failure is based on the findings of the clinical and instrumental studies.

Electrocardiography is an important tool of heart examination, which gives information on the electrical activity of the myocardium. ECG does not indicate directly structural changes in the myocardium and its contractility, but it supplies the necessary information on automaticity, excitability, and conduction of the myocardium. ECG can be used to diagnose overload and hypertrophy of various chambers of the heart. When ECG findings are compared with the clinical data, metabolic changes in the myocardium associated with hypoxia, acidosis or electrolyte disturbances can be diagnosed.

The contractile function of the myocardium can best of all be determined now by polycardiographic phase analysis of the systole of the right and left ventricles. A synchronous recording of ECG, PCG and sphygmogram of the carotid and comparison of the records help analyse the phase structure of the left ventricular systole. If a rheogram of the pulmonary artery is taken instead of the sphygmogram of the carotid artery, the phase structure of the right ventricle can be analysed. Phase analysis of the heart activity in failure is used to reveal the syndrome of cardiac hypodynamia.

Rheographic findings show indirectly the changes in the blood filling of vessels in time. Any disturbance in the heart action and condition of the vessels involves changes in the rheogram. Rheography can be used to differentiate diagnosis of affections of the right and left heart chambers. Tetrapolar rheography can also be used for calculation of stroke and minute volumes of the heart (using special formulas).

Ultrasound and echography can be used to diagnose changes in the volume of the heart chambers, thickness of their walls, and the weight of the myocardium. Computerized echocardiography and simultaneous polycardiography ensure early diagnosis of disordered phases of diastole in heart failure.

Instrumental determination of the central venous pressure helps diagnose heart failure with predominant affections of the right chambers. Differentiation between right- and left ventricular heart failures is only important for the haemodynamic form. In the pre-

sence of energy and metabolic disorders in the heart muscle, total heart failure occurs as a rule

**Left ventricular heart failure.** Affection of only the left ventricle occurs mostly in children with rheumatic heart disease, timely undiagnosed acute myocarditis, and acute nephritis. The most dangerous manifestation of the left ventricular heart failure is oedema of the lungs (see below)

**Right ventricular heart failure.** Affection of only right ventricle is a rare incidence. In most cases it combines with left-sided heart failure. It is characterized by venous engorgement (on the neck and forehead) and markedly increased venous pressure. The venous blood flow is slow. The blood from the pulmonary vessels is delivered into the vessels of the greater circulation and this often causes a marked fall in the pressure in the pulmonary vessels. If the right-sided heart failure is pronounced, the haemodynamic changes in the greater circulation are comparatively insignificant because of the large capacity of the greater circulation vessels. These changes are manifested by decreased minute volume of the heart, without involving any congestive disturbances. Congestive syndromes only develop after retention of fluids in the body and peripheral oedema that develops later due to decreased diuresis. Congestive and enlarged liver is a typical sign of right ventricular heart failure. Increased venous pressure is a characteristic sign of heart failure, of the right ventricle in particular.

Table 20 Classification of Heart Failure

| Degree          | Symptoms   |
|-----------------|--|
| H <sub>1</sub>  | Signs of circulatory insufficiency are absent at rest. Dyspnoea, pallor and weakness develop after physical strain (cry, feeding).   |
| H <sub>2A</sub> | Signs of circulatory insufficiency can be revealed at rest. Tachypnoea (not exceeding 30 per cent of normal respiratory rate). Tachycardia (10-15 per cent above normal heart rate), moderate enlargement of the heart. The liver border extends to 2-4 cm over normal.  |
| H <sub>2B</sub> | Tissues are slightly pasty. Restlessness, poor appetite, deranged sleep. Pronounced circulatory disorders. Congestion in the lesser circulation. Tachypnoea at rest (respiratory rate increased by 40-70 per cent), tachycardia (heart rate increases by 15-25 per cent), the heart sounds are dull, enlargement of the heart is significant. The liver is firm and enlarged by more than 4 cm. Periorbital oedema. Anorexia, restlessness, hyperhidrosis, vomiting. Congestive changes in the kidneys are possible. |
| H <sub>3</sub>  | Marked dyspnoea (70-100 per cent above norm), tachycardia (30-40 per cent above normal heart rate), heart sounds are dull, arrhythmia and disordered conduction. The liver is large and firm. The heart is enlarged. The child is flaccid, his appetite is very poor, oedema, ascites, oliguria.   |

Clinically the elevated venous pressure is manifested by engorgement of the jugular veins on the neck. They also pulsate with a twin positive wave. This happens because the veins do not collapse during the right ventricular systole, but become markedly engorged due to their overfilling. Engorgement of the neck veins and pressure in them can be increased by applying pressure to the liver. The hepatojugular reflex suggests insufficiency of the right heart ventricle. Overfilling with blood of the venous system combines with overfilling of the liver, spleen, vessels of subcutaneous fat, vessels of the heart and lungs. Hypoxia and prolonged blood congestion stimulate erythropoiesis, the volume of circulating blood thus increases. This increase is a compensatory response of the body, because larger volumes of blood are required to transport adequate amounts of oxygen if the blood flow is slow.

The degree of heart failure is very important to assess the patient's condition. Lang (1934) suggested that four degrees of heart failure be differentiated. This classification is now used in a slightly modified form in a paediatric cardiology (Table 20).

### General Principles of Treatment of Heart Failure

Intensive therapy in children with heart failure is aimed at eliminating or lessening hypoxia and hypoxaemia, decreasing the load on the lesser and greater circulation, improving myocardial contractility, correcting electrolyte balance, and controlling acidosis and vitamin deficiency.

**Control of hypoxia** During hypoxia the concentration in the heart muscle of macroergic phosphorus compounds and glycogen decreases, the content of lactic and pyruvic acids increases, the activity of tissue enzymes becomes inhibited, and the electrolyte balance is upset (the intracellular sodium increases while potassium decreases). Oxygen deficiency has an adverse effect on the conduction system of the heart.

Prolonged inhalations of humidified gas containing 30-40 per cent of oxygen increase oxygen tension in the myocardium, restore the decreased tissue respiration in the heart muscle, and intensify the contractile function of the myocardium. Depending on the child's age, inhalation should be conducted by various methods as described in Chapter 17.

**Lessening blood inflow to the heart** 1. Storage of blood in the lower extremities. Congestion of blood in the lower extremities can be attained by lowering the legs or by placing venous tourniquets for 20-30 minutes with subsequent gradual release of the pressure.

2. Accelerated withdrawal of liquid from the body. The lesser circulation can be unloaded by using diuretics—lasix or mannitol.

(a) a single dose of lasix for infants is 3-5 mg/kg. The administra-

tion should be repeated in 4-6 hours. The electrolytes should be controlled, (b) mannitol is an effective osmotic diuretic. It should be administered in a dose of 1 g (dry substance) per kg body weight, in the form of a 15-20 per cent solution in 20 per cent glucose or isotonic sodium chloride solution, (c) ganglioblocking agents are used in cases with markedly increased arterial pressure. A 5 per cent pentamine or a 2 per cent hexonium solution are administered slowly into the vein in a dose from 0.5 to 1.5 ml in a 40 per cent glucose solution (with permanent control of the arterial pressure).

3. Bronchodilating and spasmolytic drugs. Aminophylline is widely used now to treat cardiac failure. It has a marked cardiotonic effect and activates respiratory enzymes of the heart cells, succinyl hydrogenase and the cytochromic system. Aminophylline has a broncho-, vasodilator and diuretic effect; it stimulates contraction of the myocardium and improves haemodynamics in the lesser circulation. The preparation is administered intravenously in a dose of 1 ml of a 2.4 per cent solution per one year of age (1-2 times a day). Aminophylline is contraindicated for hypotension.

**Improvement of myocardial contractility.** Cardiac glycosides are most effective pharmacological means to treat heart failure. They have varied effect on the cardiac rhythm, myocardial contractility, on the energy, electrolyte, and hormone metabolism, and on the condition of the nervous system and the circulation regulating apparatus. When prescribing cardiac glycosides to children, the age of the child should be considered. It has been established that the concentration of cardiac glycosides in the infant myocardium increases much faster. The gap between a therapeutic and toxic dose of cardiac glycosides is very narrow. The therapeutic effect can be attained with optimum doses, smaller doses are ineffective, while bigger doses can cause poisoning. A correct dose of cardiac glycosides is the main means to decrease symptoms of heart failure.

Cardiac glycosides stimulate contraction of the heart muscle at a faster rate and with a greater force. The systole shortens and the myocardium can overcome greater resistance. Diastole elongates, the ventricles are filled with greater amount of blood, and the blood is ejected into the aorta and the pulmonary artery at a greater force. The blood flow rate and the minute volume thus increase.

Intravenous administration of strophanthin is widely used to treat acute heart failure in children. If strophanthin is contraindicated, those glycosides which are best known to the physician should be used. These may be corglycon, digoxin, celanid (isolanid), etc. Strophanthin and corglycon have the highest clearance coefficient. Their use should be preferred because of the rapid effect and easy withdrawal from the body. The full clinical effect is attained in 60-90 minutes following the intravenous administration. Strophanthin

is almost devoid of cumulative properties. Its main bulk is eliminated from the body in 6-8 hours.

Corglycon has a strophanthin-like action, but is weaker than strophanthin. The therapeutic effect of corglycon given intravenously becomes evident in 20-30 minutes and lasts from 8 to 10 hours. Corglycon is administered intravenously in 10-20 ml portions of 20-40 per cent glucose solution. Doses of cardiac glycosides are indicated in the Appendix. Cardiac glycosides should be administered slowly, during 2-5 minutes. Rapid administration (especially of strophanthin) can cause vomiting, arrhythmia (bigeminy), slow and irregular respiration, and a sudden slowing of the heart rate. Clinically glycoside poisoning in children can be manifested by anorexia, flaccidity, irritability, vomiting, and bradycardia. The ECG changes: the *S-T* wave is low, the *T* wave changes its configuration, the *Q-T* interval broadens, and the *PQ* elongates, extrasystoles appear along with disordered conduction in the atria and the ventricles.

Overdosage of strophanthin can cause maceration of the ventricles and cardiac arrest. Strophanthin poisoning occurs mostly in children treated for a long time with digitalis preparations (strophanthin action is additive to the action of the glycoside bound in the heart muscle). Cases of strophanthin overdosage should be treated by intravenous administrations of papaverine or magnesium sulphate solutions (the dose depends on age), 20-50 ml of a 2 per cent sodium citrate solution (intravenous drip), panangin, a 5 per cent unithiol solution, and plasma.

**Correction of metabolic disorders** 1. A polarizing mixture containing glucose, insulin, and potassium chloride is widely used to improve metabolism in the heart cells. This mixture facilitates penetration of potassium into the cell, stimulates induction of oxidative phosphorylation, and promotes accumulation of ATP. A polarizing mixture of the following composition can be used: 10 per cent glucose (150-200 ml), insulin 2-3 units, panangin 5-10 ml, cocarboxylase 100-200 mg, vitamins B<sub>6</sub> and C, 2 ml each.

2. In order to activate insulin, ATPase and other thiol enzymes, unithiol is administered in a dose of 5 mg/kg. Unithiol also improves tissue respiration and oxidative phosphorylation and decreases blood coagulability.

3. Vitamin B<sub>1</sub> and cocarboxylase are very important for the plasticity of heart fibres. Metabolism in the heart muscle is improved by administering vitamins B<sub>2</sub>, B<sub>6</sub>, B<sub>15</sub>, PP, sodium bicarbonate, and panangin. Panangin consists of potassium and magnesium ions and includes aspartic acid, which facilitates penetration of these ions inside the cell to improve its energy and plastic properties. The dose is 1 ml per year of age. Panangin should be administered in any affection of the heart muscle to replenish the loss of



the potassium and magnesium ions and to prevent aggravation of the condition. The preparation is produced in 10 ml ampoules containing 0.4 g anhydrous magnesium aspartate and 0.452 g anhydrous potassium aspartate. The content of one ampoule is administered slowly into the vein in emergency cases. A coated tablet of panangin contains 0.14 g anhydrous magnesium aspartate and 0.158 g anhydrous potassium aspartate. In severe cases it is recommended to take 2 tablets three times a day during a week. If the condition is of medium gravity,  $\frac{1}{2}$ -1 tablet can be taken three times a day. Contraindications for panangin are acute and chronic renal failure and hyperkalaemia.

### *Lung Oedema*

Oedema of the lungs can occur in many diseases leading to failure of the left chambers of the heart, such as severe confluent pneumonia, bronchial asthma, anaphylactic shock, comatose conditions, tumours of the brain, poisoning with organic phosphorus compounds, injuries of the head or chest, intracranial haemorrhage, epilepsy, renal failure, etc.

*Pathogenesis* Pathogenesis of lung oedema is a disputable problem. But all cases are characterized by accumulation of excess fluid in the lungs due to increased transudation. The latter can be caused by 1—increased blood pressure in the pulmonary capillaries, 2—increased vascular permeability in the lesser circulation, 3—decreased oncotic and osmotic pressure of blood, 4—increased filtration area, 5—increased water-binding capacity of the pulmonary tissue, 6—decreased filtration counter-pressure in the lungs, and 7—upset lymph outflow.

Lung oedema can be intensified by a—pronounced and long-standing hypoxia, b—hyperexcitation of the sympathetic part of the vegetative nervous system, c—upset electrolyte metabolism with retention of sodium in the pulmonary tissue, and d—increased volume of circulating blood.

Oedema of the lungs associated with cardiovascular pathology is explained by disagreement between the inflow and outflow of blood from the lesser circulation, which is accompanied by elevation of the capillary pressure and increased filtration area. The cause of toxic oedema is the breakdown of integrity of the capillary walls by poison.

The precursors of allergic lung oedema are allergic affections of the pulmonary vessels and increased content of histamine and serotonin.

Increased blood pressure in the lesser circulation capillaries upsets the gas exchange between blood and atmospheric air. Hypoxia develops progressively and this in turn increases vascular permea-

bility to a still greater extent. Due to the prevalence of hydrostatic pressure in the pulmonary capillaries over oncotic pressure, the fluid rich in proteins exudates into the alveoli. When mixed with air, the fluid foams and fills the lumen of the alveoli to interfere with gas exchange, thus intensifying hypoxia. This promotes a rapid oedema of the lungs.

*Clinic* Lung oedema can run a fulminating course, but in some cases it can develop during several days and have relapses. An attack of oedema often occurs during the night due to decreased heart rate and relatively increased blood supply to the lungs. The patient wakes up, sits in his bed, and is frightened in anticipation of an oncoming attack of asphyxia. This condition is attended by a hormonal stress and increased secretion of epinephrine and norepinephrine into the blood, and also by the release of histamine (from compounds in which it is bound). This all increases the spasm and permeability of the vessels. Asphyxia is followed by liberation of foaming sputum, which is usually pink or yellowish due to the presence of blood and large amount of plasma protein. Tachypnoea and coughing increase, respiration becomes gurgling (heard at a distance), cyanosis intensifies, and a marked tachycardia develops. Ample foamy sputum intensifies asphyxia, thus aggravating symptoms of acute heart failure.

Auscultation of the lungs reveals numerous moist rales of various calibers, which interfere with auscultation of the heart, the pulse is fast and low. Arterial pressure depends on the cause of lung oedema and competence of the heart muscle. The arterial pressure in patients with decompensated cardiac activity decreases, it increases in the absence of decompensation.

X-ray studies of patients with lung oedema reveal symmetrical cloud-like shadows, with the highest opacity in the zone of the roots. These shadows are continuous with the shadows of the roots and give the picture of butterfly wings.

*Intensive therapy of lung oedema* Despite the high variability of forms of lung oedema, the main methods of pathogenetic and symptomatic treatment are applicable practically to all cases. Complex treatment should be begun immediately in the presence of the first suspicions for oedema. The treatment should be aimed at (a) restoring patency of the airways and control of hypoxia, (b) decreasing load on the lesser circulation, (c) improving contractile function of the myocardium, (d) lessening the increased permeability of the pulmonary membrane, (e) increasing osmotic properties of the blood and forcing diuresis, (f) correcting electrolyte balance, controlling acidosis, vitamin deficiency and hyperthermia.

*Restoration of patency of airways* 1. Excess fluid should be removed from the airways by aspiration, a piece of gauze can be used to remove mucus from the mouth.

2 The quickly foaming oedematous fluid containing proteins has low surface tension and increases rapidly the amount of mucus provoking asphyxia. Alcohol vapour decreases foaming. There are several methods by which alcohol vapour inhalation can be conducted. The simplest method is by means of Bobrov's apparatus. When treating infants, a 30-75 per cent alcohol should be used (96 per cent alcohol for older children). The apparatus is connected to an oxygen cylinder.

3 Nasal tubes or masks are used with children. Oxygen is delivered at a rate of 2-3 l/min, which corresponds approximately to its 25-35 per cent concentration. To eliminate hypoxaemia the oxygen flow-rate should be increased to 10-12 l/min in the presence of marked oedema of the lungs. Oxygen with alcohol should be inhaled for 30-40 minutes, inhalations should follow at 10-15 minute intervals, during which the patient should breathe oxygen alone. This prevents excessive absorption of alcohol. If inhalation of oxygen and alcohol is carried out at an intensive therapy unit, any anaesthesia apparatus can be used. Alcohol should be placed in the vaporizer.

An organosilicon polymer is widely used as an antifoaming agent. After 2-4 minutes of inhalation of the antifoam preparation, the foaming and gurgling stop and the amount of moist rales in the lungs and cyanosis decrease.

*Decreasing venous flow to the right ventricle.* This lessens the load on the lesser circulation.

1 The venous flow is decreased by placing tourniquets on the lower extremities for 20-30 minutes (with a subsequent gradual release of the pressure on the veins). The head end of the bed should be elevated carefully.

2 Diuretics are widely used. (a) furosemide (lasix) is now very popular. The daily dose for infants is 3-5 mg/kg. In oedema of medium gravity in older children (or if an attack is mild), the dose of 20 mg is recommended for a single intake. If this is ineffective the dose should be doubled or even tripled in 15-20 minutes. A furosemide ampoule contains 2 ml of a 1 per cent solution, i.e. 20 mg. The initial dose is 10 mg, if it fails, the dose should be increased to 40-60 mg. To prevent hypokalaemia, potassium chloride should be administered intravenously (see Chapter 25). Lasix is effective in patients with increased or normal arterial pressure. In patients with hypotension lasix does not always effectively cause sufficient diuresis (due to decreased glomerular filtration),

(b) novurit is now widely used as a mercury diuretic. It is given in fractional doses (0.1-0.5 ml, during 4-5 days). The diuretic effect is seen in 30-40 minutes following the injection. The preparation is contraindicated for inflammatory changes in the kidneys,

(c) 15- and 30 per cent sterile urea solutions (0.5-1 g/kg) are used. Lyophilized solution should be preferred. Urea causes considerable

diuresis and the bladder should therefore be first catheterized. The maximum effect is attained 60 minutes after the injection, the diuretic effect lasts from 3 to 10 hours. A single injection of urea does not cause considerable changes in the electrolytes of plasma but the changes may occur during repeated urea administrations,

(d) mannitol is a strong dehydrating agent, it is less toxic, is not involved in metabolism, is quickly distributed in the intercellular space of the bodily tissues, and rapidly withdrawn from the body. The dose for children is 1 g (as dry substance) per kg body weight (as a 15-20 per cent solution in 20 per cent glucose solution or in an isotonic sodium chloride solution). The preparation is administered by drops, at a rate of 10-20 drops per minute. Intravenous administration of hypertonic mannitol solution accelerates moderately the sodium withdrawal by the kidneys, excretion of potassium with the urine also slightly increases. But osmotic diuretics (urea and mannitol) should be administered with care and appropriate control because during the first stage of their action the volume of circulating blood increases along with increasing load on the affected heart,

(e) intravenous administration of 2.4 per cent euphylline solution (from 5 to 10 ml in 10-15 ml of a 40 per cent glucose solution) is also effective in control of lung oedema,

(f) osmotic properties of blood can be increased by administering hypertonic glucose solution (20-40 per cent). While increasing the osmotic properties of blood, hypertonic solutions resist the transition of fluids from the blood vessels into the tissues and the alveoli. They also have diuretic properties. Vasodilating preparations (e.g. phentolamine, nitroglycerine, sodium nitroprusside) act directly on the peripheral vessels and are quite effective for lung oedema, ganglioblocking agents (hexonium, pentamine, arfonad) are also efficacious. They dilate the arterioles and capillaries and retain a considerable amount of blood in the peripheral vessels to promote blood redistribution between the lesser and greater circulation. This therapeutic method is called 'bloodless blood-letting'.

Sodium nitroprusside is the most popular preparation of the first group. Its effect is rapid, it is easy to batch, and its half-life period is short. Sodium nitroprusside is administered into the vein at a rate of 0.5-5.0 µg/kg per min.

Arfonad is considered to be the best in the second group. Its main advantage over the other preparations is the short half-life period. Arfonad is intended for older children. Ganglioblockers are rarely used in infants because it is difficult to calculate proper doses. Arfonad is administered by intravenous drip (100 mg per 200 ml of isotonic sodium chloride solution). The effect is obvious in 3-5 minutes after the pressure begins decreasing. In order to pre-

vent orthostatic collapse during administration of arfonad, the head end of the bed should be lowered

Vasodilating and ganglioblocking preparations should be administered with a constant control of arterial pressure. Systolic pressure should not decrease by more than 25 per cent. If the pressure fall is greater, the administration of the preparation should be discontinued and vasopressor preparations, such as norepinephrine or mesaton, should be injected intravenously in doses to 1 ml of a 0.1 per cent solution.

Diseases of the liver and kidneys associated with their dysfunction, and also severe circulatory failure are contraindications for controlled hypotension. Ganglioblocking preparations should be administered with great care to patients with mitral stenosis.

*Decreasing vascular permeability* Calcium gluconate, vitamins P and C, and also nicotinamide are used for the purpose.

The preparations of the phenothiazine series have a regulatory effect on capillary permeability and remove pathological reflexes in the lesser circulation vessels, they also have a sedative effect and can decrease pressure in the lesser and greater circulation. The use of aminazine is not however indicated for patients with normal or decreased arterial pressure because of its marked hypotensive effect. A lytic mixture containing derivatives of the phenothiazine series (aminazine) and antihistaminic preparations (pipolphen, suprastin, diphenhydramine) in 20 ml of a 40 per cent glucose solution should be administered intravenously in lung oedema (0.1 ml per year of age). The lytic mixture contains a 1 per cent promedol solution, which is given in a dose of 0.1 ml per year of age. The mixture should be administered slowly in the course of 5-10 minutes, under constant control of arterial pressure. In order to decrease the hypotensive effect of the mixture, it should be administered in 100 ml of a 10 per cent glucose solution.

Older children should be given the same doses as adults: 1 ml of a 2.5 per cent pipolphen, 1 ml of a 1 per cent promedol solution, 0.2-0.5 ml of a 2.5 per cent aminazine solution with an additive of 0.5 ml of a 0.05 per cent strophanthin solution. In several minutes which follow after the administration of this mixture, dyspnoea and cyanosis decrease, the amount of moist rales in the lungs diminishes, the pulse slows down, and the patient falls asleep due to a strong sedative effect of the mixture.

Corticosteroids normalize pathologically increased vascular permeability. Moreover, adrenocortical insufficiency develops in severe cardiovascular pathologies. This, in turn, causes a fall of the arterial pressure. Prednisolone is administered intravenously to children in a dose from 1 to 2 mg/kg, irrespective of age. If the attack is of moderate gravity,  $\frac{1}{2}$  daily dose is administered by a single injection, while the remaining quantity should be given per os.

If the arterial pressure is low, the daily dose should be administered during the course of 90-120 minutes, in 7 5-15 mg portions, at 20-30-minute intervals

If oedema of the lung is due to left ventricular heart failure, an important measure is *intravenous administration of cardiac glycosides to maintain contractile function of the myocardium*. Strophanthin is the most efficacious substance for acute heart failure. With intravenous administration its effect is practically immediate and persists for 7-8 hours. Strophanthin increases systole and the minute volume, it decreases venous pressure, and the heart becomes able to intensify its contractions in response to increasing load, the blood flow is also accelerated. Strophanthin is usually administered in a dose of 0.5 ml of a 0.05 per cent solution in a 40 per cent glucose solution. A single strophanthin dose for infants is 0.01 mg/kg, or 0.02 ml of a 0.05 per cent solution per kg body weight.

*Correction of acidosis* This is attained by intravenous administration of a 4 per cent sodium hydrocarbonate solution (2-4 ml/kg)

### ACUTE VASCULAR INSUFFICIENCY

Vascular insufficiency occurs in cases where the balance between the circulating blood volume and the capacity of the vascular system is upset

*Clinical picture and differential diagnosis* Treatment of vascular insufficiency differs from treatment of heart failure, differentiation between these pathologies is therefore necessary to ensure proper treatment. The patient with heart failure prefers an elevated posture because the volume of circulating blood increases in the horizontal position to intensify dyspnoea. When the patient lies, the muscles of the shoulder girdle are not involved in the respiratory act. The horizontal position of patients with vascular insufficiency is more physiologically comfortable because the circulating blood volume increases and the blood supply to the brain is improved. The skin of a patient with heart failure is cyanotic (mostly acrocyanotic), while in vascular failure it is pallid and covered with cold sweat. The neck veins of a patient with heart failure are engorged, while in patients with vascular failure they are empty and invisible. Arterial pressure falls and venous increases in heart failure, while in vascular failure both the arterial and venous pressures are low. The heart is enlarged in cases with acute heart failure and the gallop rhythm is not infrequent. The dimensions of the heart in vascular failure remain normal and the gallop rhythm is absent. Heart failure is characterized by congestion in the lungs and the liver, while these symptoms are absent in vascular failure.

When applying differential diagnosis it is necessary to remember that acute heart failure may combine with vascular insufficiency

Table 21 gives comparative characteristics of acute heart and acute vascular failures. Vascular failure manifests as syncope, collapse and shock.

Table 21 Differential Diagnosis of Acute Heart Failure and Acute Vascular Insufficiency

| Physiological parameters       | Heart failure   | Vascular failure   |
|--------------------------------|---|--|
| Characteristic position in bed | Elevated  | Horizontal   |
| Peripheral veins               | Engorged (neck veins pulsate)   | Collapsed  |
| Skin                           | Cyanotic, mostly acrocyanotic   | Pallid and sweaty<br>Diffuse cyanosis is frequent, grey cyanosis |
| Cardiac dullness               | Enlarged  | Normal or diminished   |
| Liver                          | Enlarged  | Usually normal   |
| Congestion in lungs            | Present   | Absent   |
| Respiration                    | Accelerated, often intense and difficult                                    | Accelerated, shallow, easy                                       |
| Arterial pressure              | May be decreased (mostly systolic), sometimes increased, pulse pressure low | Always decreased (mostly diastolic)                              |
| Venous pressure                | Increased   | Decreased  |
| Circulating blood volume       | Increased   | Decreased  |

*Intensive therapy of acute vascular failure* The complex treatment should be aimed at the following (a) restoring the circulating blood volume, (b) improving microcirculation, (c) decreasing reflex impulsion associated with injury in the vascular wall, (d) elimination of acidosis and metabolic disorders. The therapy depends on the degree of vascular insufficiency and the stage of shock. Spasm of arterioles occurs at the first stage, owing to which a considerable part of capillaries is excluded from the circulation, which is a compensatory reaction of the body. Arterial pressure remains normal because the decreased volume of circulating blood is sufficient for filling the decreased capacity of the vessels, but the organs and tissues suffer from insufficient blood flow-rate. Vasoconstricting preparations are contraindicated in such cases. The first measure to be taken before the vessels are affected by paralysis is restoration of the circulating blood volume.

Haematocrit and the central venous pressure should be controlled in massive blood transfusions. They characterize efficacy of the therapy and the load on the heart. Increased central venous pressure indicates overload on the right chambers of the heart because of the increased blood inflow to them. High haematocrit and low central venous pressure indicate sufficient amounts of transfused blood and fluids.

Low arterial pressure indicates weakened compensatory mechanisms and paralysis of the vessels. In such cases, along with blood transfusion it is necessary to administer vasoconstricting substances. It should be remembered that no medicamentous therapy can compensate for the deficient amount of fluids.

Analgesics, antihistaminics and sedatives can be indicated for acute vascular failure. The diseased children need oxygen therapy because hypoxia and hypoxaemia develop due to decreased blood flow-rate.

### Syncope

This is a mild form of acute vascular failure with a short-lasting unconsciousness caused by a transient cerebral ischaemia.

*Aetiology and pathogenesis.* A syncope is in most cases caused by an acute reflex fall in the arterial tone. Dystonia of the sympathetic nervous system and increased tone of nervus vagus, causing a considerable fall in the arterial and venous pressure, occur usually in children with increased vasomotor and emotional lability. Acute infectious diseases of the near past, debilitation of the body due to starvation or underfeeding predispose to syncopes. A syncope can occur during an injection from a skin puncture by the needle, in the sight of blood, during tooth extirpation, during an abrupt transition from the lying position to the upright posture. As a rule, the syncope occurs unexpectedly. In some cases the faint is preceded by dizziness, stars in the eyes, nausea, the feeling of heaviness and numbness in the arms and legs.

*Clinical picture.* The condition of the child is characterized by pallor of his skin and mucosa, the whites of the eyes are seen, the pupils are contracted and do not react to light, respiration is shallow and slow, the pulse is small and low, the limbs are cold, the child is covered with cold sweat. Arterial pressure is very low, the peripheral veins are empty. This condition can last from a few seconds to 2-3 minutes. The child regains consciousness, he opens the eyes, his lips begin moving, he responds to extraneous stimuli, colour begins to return to the face.

It is necessary to differentiate between a syncope and a hysterical or epileptic faint, acute heart failure, and comatose conditions. A minor epilepsy attack is characterized by a loss of consciousness for 1-2 seconds, the child suddenly becomes motionless during his game or talk, his eyes become fixed, the eyelids or face muscles begin twitching. A hysteric attack does not alter the colour of the face, the pulse is normal, the eyelids tremble. A coma is characterized by a longer loss of consciousness, grave general condition, disordered vital functions, and symptoms corresponding to a particular type of coma.



*Treatment* The child should be brought into fresh air, his collar should be undone, the waist loosened, he should be placed in the horizontal position with his legs slightly elevated over the head level. Water should be sprinkled over the chest and face, the child should be called up loudly by name and ammonia spirit given to smell. If these measures fail, caffeine, cordiamine, ephedrine or other stimulant should be injected subcutaneously in age doses.

### Arrhythmias

Arrhythmias occur more frequently in grown up children, although they may also occur in children of all ages. They arise due to disorders of the main functions of the heart muscle—automatism, excitability, conduction, and contractility. Some types of arrhythmia can cause a cardiac arrest or they can upset the function of vital organs. Children with arrhythmia should therefore be constantly observed and treated at intensive therapy units. Among such arrhythmias are paroxysmal tachycardia, fibrillation, auricular flutter, and complete atrioventricular block with the Morgagni-Adams-Stokes syndrome.

#### *Paroxysmal Tachycardia*

This is accelerated action of the heart, 2-3 times exceeding the normal rate. Paroxysmal tachycardia can be regarded as a succession of extrasystoles which follow one another at regular frequent intervals. Atrial, atrioventricular and ventricular forms of paroxysmal tachycardia are differentiated. The atrial or atrioventricular form is more common for children.

*Clinical picture* An attack of tachycardia begins suddenly. The child feels discomfort in the heart (as if a blow or a prick). The heart rate increases from 150 to 500 beats per minute, the child feels dizziness and nausea, vomiting and convulsions develop, and sometimes the child loses consciousness. The urge to urinate (at 10-15-minute intervals) is especially characteristic. Inspection of the child reveals pallor of the skin and mucosa, cyanosis of the face and other parts develops if an attack is prolonged. Heart failure is possible, the arterial pressure falls, and dyspnoea develops. The liver and the spleen are enlarged. The heart boundaries are first normal but can later broaden. Auscultation reveals short and clear heart sounds. The pendulum rhythm of the heart is characteristic.

Frequent contractions in paroxysmal tachycardia shorten the diastole and decrease the blood inflow to the heart. The minute volume decreases as well (despite the high heart rate). The blood supply to the organs and tissues is thus impaired and the oxygen absorption in tissues decreases. Almost simultaneous contraction of the atria and the ventricles accounts for incomplete emptying of the

atria during a diastole, which is short, the blood is regurgitated into the veins during atrial contractions. The neck veins are strongly engorged and the jugular veins pulsate synchronously with the atrial contractions. Venous congestion occurs in the greater and lesser circulation as a result of which pulmonary gas exchange becomes upset with the development of hypoxia and hypoxaemia.

Changes of the blood circulation are pronounced only during prolonged attacks. Even frequent but short attacks have no effect on the circulation of blood.

The atrial form of paroxysmal tachycardia is characterized by markedly shorter *T-P* interval and the *P* wave superimposes on the *T* wave to disfigure it, the ventricular complex can be normal or altered. If the *P* wave is superimposed by the *T* wave, it can be located only by comparison of ECG on termination of the attack.

During atrioventricular paroxysmal tachycardia the *P* wave depends on the localization of the ectopic focus. If it is in the atrial portion of the node, the negative *P* wave precedes the *R* wave, if it is in the middle portion the *P* and *R* waves are superimposed, and the negative *P* wave is difficult to differentiate, if the focus is in the lower portion of the node, the negative *P* wave is found between the *R* and *T* waves.

During ventricular paroxysmal tachycardia the *P* wave is absent while the ventricular complex is disfigured. In order to differentiate between the forms of paroxysmal tachycardia, it is necessary to compare ECG taken during and after the attack.

The amplitude of the first and second heart sounds increases and of the third sound decreases in the PCG taken during the attack of paroxysmal tachycardia, systolic murmur can be heard. The phonocardiographic picture normalizes on abatement of the attack.

**Treatment** In diseases of the cardiovascular system paroxysmal tachycardia impairs drastically the condition of the child, normalization of the heart rhythm becomes an urgent necessity. The attack can sometimes be arrested by reflex irritation of the nervus vagus. Pressure on the eyeball or the carotid sinus (on the right side) is applied for the purpose. These techniques are more effective for the atrial form of tachycardia. The Valsalva test can also be used: after a deep inspiration the maximum attempt at expiration is made for 10-20 seconds with the nose and mouth tightly closed.

Less effective are mechanical methods that can be performed by the patient himself: (1) slow and deep respiration, (2) keeping breath for a long time with the body in the horizontal position, (3) artificial induction of vomiting, (4) swallowing of solid food or drinking carbonated water, (5) strong pressure on the upper abdomen, (6) pressing the legs to the abdomen, (7) sponging with cold water.

Highly efficacious preparations for elimination of attacks of paroxysmal tachycardia are beta-blockers, e.g. anaprilin, obsidan,

digitalis preparations (preferably celand (celand) or digoxin, having a more pronounced vagotropic effect than strophanthin, posaconamide, verapamil (isoptin), ajmaline, and quinidine.

Anaprilin is administered in a dose of 1 mg in isotonic sodium chloride solution. The injection is slow, with permanent ECG control. The preparation can also be injected slowly into the vein (2-4 mg). If arterial hypotension, bradycardia or atrioventricular block develops, the infusion should be discontinued immediately. Beta-blocker can be administered repeatedly in 3-5 minutes, the overall dose not exceeding 1 mg. Cardiac glycosides should be administered in the above-specified dose. They are especially indicated for patients with paroxysmal tachycardia in the presence of decreased contractile power of the myocardium. Cardiac glycosides are contraindicated for supraventricular tachycardia that develops during digitalis therapy, or in cases suspected for hypokalaemia. Verapamil should be slowly injected intravenously in a dose not exceeding 5 mg; repeated injections should be made no earlier than in 20-30 minutes. Like cardiac glycosides, verapamil should not be used in cases where ventricular tachycardia or premature excitation in combination with atrial flutter and fibrillation cannot be excluded.

Ajmaline (gilurvtmal) is injected either intramuscularly or intravenously, in a dose from 15 to 30 mg. Repeated halfdoses should be injected not earlier than in 30 minutes. If the condition is not critical, it is recommended to begin therapy with intramuscular administrations of the preparation. If this measure fails, an intravenous injection should be made. Ajmaline often provokes arterial hypertension, bradycardia, atrioventricular and intraventricular block. ECG control is therefore necessary.

Novocainamide aborts attacks of paroxysmal tachycardia in about 80 per cent of cases. It is administered intravenously, by drip or slow injection (3-5 ml of a 10 per cent solution). Novocainamide can cause hypotension (to collapse). The therapy should therefore be begun with peroral administration of  $1\frac{1}{2}$  tablet 2-4 times a day. If the effect is absent, the preparation should be administered intravenously. Signs of heart failure and bundle-branch heart block are contraindications for this preparation.

Quinidine is used in cases where the child's condition does not require urgent measures, in the absence of heart failure or myocardial affection, or in the absence of intraventricular conduction. Quinidine is used in a dose of 0.1-0.2 g, at 3-hour intervals (the overall dose not exceeding 0.8-1.2 g). The heart rhythm usually normalizes after 2 or 3 intakes.

Combinations of various preparations can be used to treat paroxysmal tachycardia. Treatment with beta-blockers and digitalis preparations, whose efficacy is higher even with smaller single doses,

has been studied best of all. The absence of effect from using all mentioned means is an indication for electric impulse therapy.

### *Atrial Fibrillation*

This is the arrhythmia characterized by irregular contractions and twitchings of separate groups of the atrial muscles, which occur instead of normal atrial contractions.

*Aetiology and pathogenesis* Atrial fibrillation arises during an acute attack of active rheumatism, in some congenital heart diseases (defect of the interatrial septum, tricuspid abnormalities, interventricular septum defects), in diphtheritic and primary idiopathic myocarditis. Atrial fibrillation can occur after administration of digitalis preparations, adrenaline, acetylcholine potassium, and also during catheterization of the heart.

*Clinical picture* Depending on the contraction rate, atrial fibrillation and atrial flutter are differentiated. Flutter and fibrillation were formerly considered to be different affections. But these forms of arrhythmia can convert into one another and therefore they are now described as variations of one and the same disease. The contraction rate during atrial flutter varies between 200 and 350 per minute, while in fibrillation contractions vary between 350 and 360. The rate of ventricular contractions in such cases is 2 or 3 times lower. According to the frequency of heart contractions per minute, three forms of atrial fibrillation are distinguished: tachy-, brady-, and normo-arrhythmic. The tachy-arrhythmic form is the most common. Next follow normo-arrhythmic, and still less frequently occurs the brady-arrhythmic fibrillation.

Children with the tachy-arrhythmic form complain of pain in the heart, palpitation, bad condition, deranged sleep and appetite. Auscultation reveals systolic murmurs, heart sounds are dulled during rapid contractions. Slapping sounds follow at irregular intervals. The appearance of slapping sounds is probably associated with insufficient filling of the ventricles with blood due to a markedly shortened diastole. Many contractions of the atrium do not reach the periphery because they are generated following a very short diastole. Simultaneous counting of the heart rate (by auscultation) and of the pulse rate on the radial artery is used to determine the pulse deficit, which is a typical clinical manifestation of this form of arrhythmia.

The main ECG characteristics are the absence of the *P* wave or appearance of many very small *P* waves (in compliance with the fibrillation rate) and irregular spacing of ventricular complexes. The ventricular complexes are usually disfigured due to superimposition of the atrial waves or heart affections. The tachy-arrhythmic form of atrial fibrillation causes congestion in the lesser circulation and heart failure.

The brady-arrhythmic form of atrial fibrillation is characterized by the heart rate not exceeding 80-90 per minute. The diagnosis of this form is difficult because the heart rate is nearly normal. Diagnosis is established by thoroughly auscultating the heart and counting the pulse rate. The difference in the force and irregularity of the pulse waves are less distinct than in tachy-arrhythmic fibrillation. Pulse deficit is small or absent. ECG helps establish the diagnosis: the *P* wave is absent and small waves can only be seen. The ventricular complex remains normal but the intervals between the *QRS* complexes are irregular. The pulse wave and the incisure on the carotid curve of a sphygmogram are absent, the amplitude of the pulse wave on the radial artery curve is low; secondary waves can be seen.

The normo-arrhythmic form of atrial fibrillation occurs suddenly, after a strong emotional stress, and can develop in practically healthy children; it can end as suddenly as it began, but in some cases it may convert into a permanent condition.

The tachy-arrhythmic fibrillation should be differentiated from polytopic group extrasystoles. A correct diagnosis can be established by means of ECG.

It is more difficult to diagnose brady-arrhythmic atrial fibrillation. It can be mistaken for respiratory arrhythmia or sinus bradycardia. Respiratory arrhythmia disappears when the patient holds his breath. Physical exercise removes sinus bradycardia, while the brady-arrhythmic form of fibrillation converts into the tachy-arrhythmic form during exercise.

*Treatment* Digitalis preparations, quinidine and quinine are used to treat atrial fibrillation. Digitalis has proved to be the most effective; it acts on the centre of the nervus vagus and its endings to slow down the conduction of the impulse, thus causing retardation of the heart rate. Digitalis increases the tone of the nervus vagus, sharply decreasing excitability of the atrioventricular node. This, in turn, becomes less responsive to the multitude of impulses delivered from the fibrillating atria. The heart rate is slowed, the diastole increases, systole intensifies, and the pulse deficit disappears.

Big doses of digitalis can intensify atrial fibrillation; children over 7 years old should be given 0.05 g while infants only 0.03 g (three times a day). Digitalis should be administered till the therapeutic effect becomes obvious: the rate of ventricular contractions decreases to 70-80 per minute. The preparation can then be suspended. If digitalis fails to give the desired effect, beta-blockers should also be administered (per os, in moderate doses). Verapamil can be given in addition to digitalis and beta-blockers. This combination is efficacious in severe cases when tachy-arrhythmia is persistent.

Restoration of normal rhythm after brady-arrhythmia is attained only by diuretics or novocainamide and quinidine. Novocainamide

is effective for paroxysmal atrial fibrillation, while its efficacy is low against tachy- and brady-arrhythmias. Quinidine is more indicated for brady-arrhythmia. The preparation should be administered per os in 0.1-0.2 g doses, first once a day and later (in 3-4 days) this dose should be given 2-3 times a day. If no effect is attained, quinidine may be given in repeated courses.

Defibrillators are now widely used to treat atrial fibrillation. Defibrillation is performed with surface anaesthesia, with voltages in the range of 1000-4000 V, impulses lasting for 0.01 second. Electric discharges stimulate all fibres of the myocardium simultaneously, eliminate asynchronism of excitation and normalize conduction.

### *Atrial Flutter*

The frequency of atrial flutter varies between 250 and 350 per minute, while ventricular contractions follow at a rate not exceeding 150-180 per minute.

*Aetiology* Atrial flutter occurs in children markedly less frequently than fibrillation. It occurs in rheumatic myocarditis in the active stage of the disease, in some congenital heart diseases associated with significant dilatation of the atria and increased pressure in the lesser circulation. Several *P* waves can be seen in ECG during each cardiac cycle. The size and shape of the *P* wave are constant and the waves are evenly spaced. The *QRS* ventricular complex remains unchanged, but in some cases the *P* wave is superimposed on the *P* and *T* waves.

A correct diagnosis is often established by ECG. Treatment is the same as for atrial fibrillation.

### *Complete Atrioventricular Block with Morgagni-Adams-Stokes Syndrome*

A complete atrioventricular block occurs in upset conduction from the atria to the ventricles and they contract independently of each other. The atria contract from impulses sent from the sinus node, while contractions of the ventricles are stimulated by impulses arising in the ventricles themselves. The rate of atrial contractions is almost normal, while the ventricles contract about two times slower.

Conduction in the atrioventricular node, or His' bundle, is disturbed due to development of a persistent and strong focus of excitation (parabiosis). The parabiotic part of the conduction system in neurogenic block inhibits transmission of the excitation wave from the sinus node to the ventricle.

*Aetiology* Complete atrioventricular block can be caused by diphtheritic myocarditis, rheumocarditis, scarlet fever, sepsis, and congenital heart diseases. Congenital block is a result of abnormal growth of the conduction system in the region of the atrioventricu-

lar node and His' bundle As impulses arise in this node, the ventricles contract at a rate of 40-50 per minute If the site of affection is lower, the impulses arise in the bundle at the point of its ramification into the left and right branch, the ventricles then contract at a rate of 20-30 per minute

If the rate of contractions is 40-50 per minute, the cardiovascular system of the child adapts itself to the slow rhythm Systole increases (with respect to the blood volume), the ventricles eject larger amounts of blood, and the myocardium becomes hypertrophied

*Clinical picture and diagnosis* Slowing the heart rate to 20-30 per minute causes anaemization of the brain and development of the Morgagni-Adams-Stokes syndrome The patient feels a sudden discomfort, dizziness, and then faints The face first reddens and then becomes deadly pale with a cyanotic hue The neck veins become engorged, the pulse is impalpable, and the respiration is deep The facial muscles begin twitching, the extremities jerk, and the patient defaecates and urinates involuntarily The attack may continue from a few seconds to 2 minutes If the attack is longer or the heart's action is not restored, the patient dies After restoration of the cardiac activity, the child regains consciousness

The *P* wave and the ventricular complex *QRS* are located on ECG independently of one another The number of *P* waves is much greater than of the ventricular complexes, and they are usually spaced evenly The ventricular complexes are also regularly spaced, but the *P-P* interval is much longer The *P* wave can be seen in various regions of the ECG it can precede the *QRS* complex be superimposed on it, or follow next to it The configuration of the ventricular complex is normal if the source of rhythmic impulses is found in His' bundle above its bifurcation point If the source is located in either of the branches, the *QRS* complex changes its configuration it broadens, a serrated pattern appears on the *P* wave, while the *T* wave becomes negative

The diagnosis should be differentiated from sinus bradycardia or incomplete atrioventricular block A markedly slowed pulse is not characteristic of sinus bradycardia Sinus bradycardia can temporarily be eliminated by exercise or atropine, while the pulse does not change in complete block Incomplete block can convert into complete during physical strain ECG should be used in all dubious cases

*Treatment* This should be pathogenetic Depending on age, from 0.1 to 0.5 ml of a 0.1 per cent atropine solution should be injected subcutaneously (or 0.1 per cent adrenaline solution) Corticosteroids are also effective in non-persistent atrioventricular block Congenital atrioventricular block needs no special treatment Attacks of the Morgagni-Adams-Stokes disease should be treated with subcutaneous injections of adrenaline (0.3-0.5 ml) or ephedrine ( $1\frac{1}{2}$  tablet, 2-3 times a day) Complete block associated with attacks of the Morgagni-

Adams-Stokes disease should be managed by means of cardiostimulators, which can stimulate the heart through the closed chest at the desired rate

## Chapter 19

### Intensive Therapy of Shock

Shock is an extremely severe critical condition characterized by pronounced depression of the central nervous system, severe central and peripheral circulatory disorders and marked respiratory and metabolic disturbances. These changes as a rule, arise in response to extraordinarily strong stimuli caused by endo- and exogenic factors. In rare cases an adult may develop a shock in response to a common stimulus, but his initial condition must be grave and the reactivity of the body extraordinarily high.

The specific features of pathogenesis, clinical course and therapy of shock in children are associated with a relative immaturity of some organs and systems. These are especially pronounced in neonates and become levelled with age. The cerebral function of neonates and infants of the first months of life is vulnerable to extraneous stimuli and the vital functions are mainly regulated by subcortical structures—the diencephalon and the thalamopallidal region. In these conditions the cerebral cortex has no significant effect on the physiological functions. Pain and other strong stimuli are not yet localized; the coordination of the conjugated regions is absent. Even comparatively weak stimuli (pain, hyperthermia, intoxication, and the like) can therefore cause generalized excitation of the central nervous system with subsequent inhibition and development of shock. Immaturity of the nervous structures regulating circulation of blood, respiration, reactivity to pain, intoxication, and other stimuli accounts for the rapid onset of severe circulatory and gas-exchange disturbances in infants.

*Aetiology and classification.* There exist many aetiological factors which can provoke shock: bleeding, dehydration, injury, burn, electricity, cold, infection, allergic response to various medicines, blood, proteins, severe heart failure, and other conditions.

The following types of shock commonly occur in paediatric practice:

- 1 Haemorrhagic (hypovolaemic) shock. It develops due to profuse bleeding and strong dehydration of the body.

- 2 Traumatic shock. This can be caused by severe trauma, operative injury, burn, exposure to electricity or very low temperature, compression of the body.

- 3 Septic shock. This type of shock arises in severe infections and septic conditions. Gram-negative flora has been recently found to be responsible for septic shock.



4 Anaphylactic shock This can be caused by an allergic response to administration of antibiotics, transfusion of protein preparations sera, vaccines, and the like

In addition to the above classification based on the aetiological factors, distinguished also are various phases or stages of shock -erectile, torpid, and terminal Reversible and irreversible types of shock are sometimes differentiated

### GENERAL PRINCIPLES OF INTENSIVE THERAPY OF SHOCK

Depending on the type of shock, intensive therapy may have some specific features The general principle is a complex pathogenetic therapy aimed at eliminating the current of impulses into the central

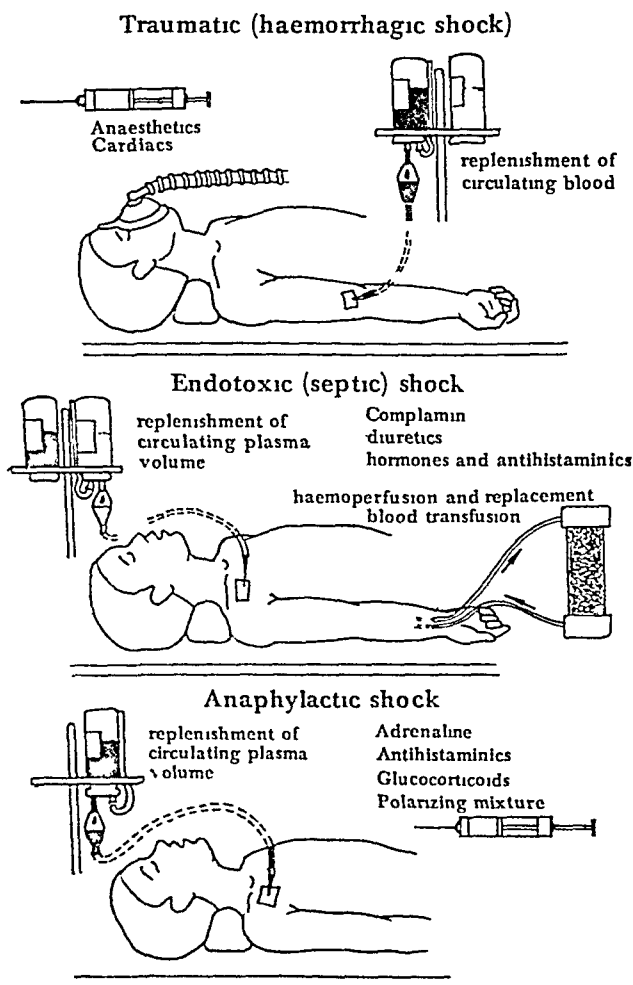


Fig 50 Intensive therapy of shock

nervous system and the associated disturbances in the nervous activity, at eradicating circulatory disorders and oxygen deficit, at normalizing the main metabolic processes (Fig 50) and eliminating rheological disorders

The result of anti-shock therapy depends on its timely application, which can be attained by a correct organization of the resuscitation service. All shock patients should be given the necessary treatment without delay and in a definite order. Every department intended to render urgent aid should always be able to give anaesthesia by means of special apparatuses. Sterile systems for intravenous infusions, laryngoscopes and endotracheal tubes, catheters, syringes with needles, electric aspirators for removal of mucus from the airways should always be prepared for immediate use.

Shock patients should be placed in the resuscitation unit or operative room without giving them any examination or sanitary treatment. All diagnostic and medical manipulations should be carried out simultaneously on the operation table. Clothes should be carefully removed from the victim. His condition and the blood group should be determined. The subclavian vein should be punctured and catheterized according to Seldinger (or venesection should be done). The system for fluid transfusions should be prepared for use.

## INTENSIVE THERAPY OF VARIOUS TYPES OF SHOCK

### Haemorrhagic (Hypovolaemic) Shock

*Pathogenesis.* A considerable blood loss or other body fluid (hypovolaemia) first of all decreases the cardiac output. The compensatory passage of liquid from the pre-capillary and even intracellular space into the capillaries lessens the danger only for a short early period. In response to the decreased cardiac output, the tone of the veins (and then arteries) increases by reflex. Blood circulation in the vital organs (brain, myocardium, kidneys) is thus maintained. If hypovolaemia is not corrected, the spasm of arterioles, pre- and post-capillary sphincters increases to slow down the blood flow-rate and to impair the rheological properties of blood. The formed blood elements clog together, the red cells aggregate (sludging), and blood is retained in the microcirculatory system. The pre-capillary sphincters are then paralysed to cause stasis, thus intensifying the hydrostatic pressure, increasing the capillary permeability, and causing oedema of tissues. All this upsets metabolism and nutrition of tissues. A local and then generalized metabolic acidosis develops. Acidosis impairs the myocardial function. Oxygenation of tissues is also drastically impaired when direct arteriovenous anastomoses open at the moment of spasm of the pre- and post-capillary sphinc-

ters and part of arterial blood is directed into the veins by passing tissues

Parallel to the described mechanisms, the increased activation of the sympathoadrenal system (which increases the vascular tone and the heart rate) increases also the oxygen demand. The vicious circle is thus formed: tissues require more oxygen but the oxygen supply is decreased. This upsets their function.

Marked disturbances in the respiratory and other functions of the lungs, morphological changes (thrombus formation, oedema, atelectasis, fat embolism), which arise during shock, have made it possible to regard this condition as a specific nosological form called 'shock lung'. A similar picture is observed in renal pathology ('shock kidney').

*Clinical picture* Manifestations of haemorrhagic shock in children depend on the stage. Stage I shock in children is marked by anxiety, moderate pallor and tachycardia. Arterial pressure can be normal and even slightly elevated. The central venous pressure tends to a moderate increase. Respiration is slightly accelerated, and respiratory alkalosis is possible. Blood loss and hypovolaemia may vary between 15 and 20 per cent of the circulating blood volume. Stage II shock is characterized by stupor, more pronounced pallor, and acrocyanosis. Dyspnoea is marked. Arterial and central venous pressure fall. The blood thickens (increased haematocrit), metabolic acidosis develops. Blood loss and hypovolaemia may reach 25-40 per cent of the circulating blood volume. During the third stage the patient is unconscious, pallor is deadly. Respiration is very fast and shallow; the arterial pressure falls by more than 50-60 per cent of initial. Tachycardia is significant, the pulse is weak. Anuria develops. The blood is characterized by a marked respiratory and metabolic acidosis. Blood loss exceeds 40 per cent of the circulating volume. The fourth stage is similar to agony.

*Intensive therapy* This begins with replenishment of the lost fluid (normalization of the volaemic status). Urgent infusion therapy begins with injection of polyglucin (20-30 ml/kg) with hydrocortisone (25-30 mg/kg) and vitamin C (3-5 ml of a 5 per cent solution) until the systolic arterial pressure rises to 80-90 mm Hg, the infusion should then be conducted by drip. Rheopolyglucin should then be infused in the same quantity, which normalizes microcirculation by eliminating red cell aggregation and returning them into the circulation system. Transfusion of dextran preparations ensures rapid replenishment of the circulating blood volume with moderate dilution, which improves the rheological properties of blood, systemic haemodynamics, and microcirculation. All this enables the physicians to determine the blood group and the Rhesus factor without haste and to proceed to transfusion of blood (better freshly heparinized or at least fresh citrate blood). It is not recommended

to use blood older than 3 days for children. If there was no bleeding, blood substitutes can only be transfused. But the proportion of blood should not exceed 50-60 per cent of the transfused fluid even in the presence of blood loss, because massive blood transfusions can cause severe complications. After blood transfusion it is recommended to administer haemodes (10-12 ml/kg), which has detoxicating properties, improves microcirculation, and increases diuresis.

Infusion therapy is combined with administration of some pharmacological preparations, which ensure vegetative protection and removal of various dysfunctions of vital organs and systems.

During stabilization of arterial pressure it is recommended to administer droperidol (0.1 ml/kg) to remove vasoconstriction and to ensure effective neurovegetative protection. Droperidol also decreases sensitivity to catecholamines and inhibits motor activity. Promedol and nitrous oxide, in combination with oxygen, should be used for analgesia, if necessary.

In the absence of danger of haemorrhage, heparin should obligatorily be used (20-30 U/kg). It should be administered at 3-4-hour intervals with coagulographic control (or with Lee-White time control). Heparin prevents intravascular coagulation, improves microcirculation, and is an effective antihypoxant and protector of cell membranes.

It is also reasonable to administer intravenously trasyolol or similar preparations (contrykal, gordox), which inhibit the kallikrein-kinin system whose activity increases markedly in shock.

Simultaneously it is necessary to carry out therapy aimed at elimination of respiratory insufficiency (cleaning airways, inhalation of oxygen through nasal tubes, and spontaneous respiration with constant positive pressure). In the presence of pronounced hypercapnia, hypoxaemia, or respiratory arrhythmia, the patient should be intubated for artificial lung ventilation until gas exchange is stabilized and the general condition improved. The main criteria by which the efficacy of the conducted therapy can be assessed are the arterial pressure (age norms), normalized microcirculation (disappearance of pallor, cyanosis, or marbling, and restoration of colour of the skin), and normal diuresis.

Shock is in most cases the complication of various pathological conditions, which require urgent surgical treatment. Therefore, a question of possible operation and optimum anaesthesia conditions arises. It is recommended to postpone operation until blood circulation is stabilized and shock is managed. But in critical conditions (continuing bleeding), the operation should be begun simultaneously with antishock therapy. If bleeding is moderate, operative intervention may be postponed for 20-25 minutes in order to normalize gas exchange and to conduct correcting infusion therapy, which will minimize the risk of anaesthesia and operation.

## Traumatic Shock

A common aetiological factor is a trauma or pain syndrome. But these factors are usually combined with blood loss.

*Pathogenesis* Traumatic shock develops like haemorrhagic shock in many respects. But the triggering mechanism of microcirculatory disturbances in severe injuries is a current of pain impulses. The specific character of genesis of shock depends on the character of trauma: respiratory distress in injuries of thoracic organs, the danger of fat embolism in fractures of tubular bones, etc.

*Clinical picture* The course of traumatic shock in a child differs from that in an adult: the erectile stage is not always quite obvious. At the same time, in the absence of profuse bleeding, the arterial pressure can be stable for longer periods of time. The respiratory function is however depressed in children sooner than in adults.

*Intensive therapy* The therapy of traumatic shock usually begins with treatment of the pain syndrome. Analgesia and block of pathological reflexes prevent spasms in the microcirculatory system.

Depending on the character of injury, analgesia can be local or general. Procaine is very effective in many cases (see Ch. 13). Epidural anaesthesia is very efficacious but it can only be used in the presence of stable arterial pressure. Central analgesics are also very effective, but they cannot be used in injuries of the abdominal organs, in the absence of a verified diagnosis because central analgesia can blur the clinical picture. It should also be remembered that central analgesics act on the respiratory centre and can cause respiratory distress. It is therefore necessary to be prepared to manage respiratory disorders when using central analgesics. It is recommended that ketamine should be used in traumatic shock along with neuroleptanalgesics because, in addition to the analgesic effect, they also remove microcirculatory spasms.

General anaesthetics are often used to remove pain syndrome, e.g. nitrous oxide and oxygen in the ratio of 2 : 1 or even 1 : 1. Sodium oxybate is administered intravenously in a dose of 70-100 mg per kg body weight. The daily dose can be increased to 400-600 mg/kg.

Infusion therapy is the same as in haemorrhagic shock (including replenishment of the lost liquid and improvement of the rheological properties of blood).

A very important medical factor is operation, the effective method of correcting disorders and arresting haemorrhage. Therefore, in the presence of bleeding, the only contraindication for operation is the 4th stage shock (Anaesthesia in shock was discussed in Ch. 19).

## Burn Shock

Burn shock is caused by extraordinarily severe pain with large wound areas and toxæmia. The *clinical picture* of burn shock is characterized by a more pronounced and prolonged (to several hours) erectile phase. The gravity of patient's condition is during this period masked by euphoria, absence of hypotension and excitement. This can mislead the physician and result in incorrect urgent aid. All antishock measures should be taken, otherwise the burn shock converts into its torpid phase. This is characterized by all symptoms of a severe shock caused by mechanical injury, but attended by more pronounced renal failure (to anuria) because of the absorption of great amount of toxins and products of decomposition from the vast burn surface.

During this period the general principles of burn shock *therapy* do not differ from the above-described therapy. Special attention should be paid to the treatment of renal failure. Procaine paraneurial block and warmth to the lumbar region should be applied. Diaphylline, euphylline and lasix should also be administered. Acidosis should be controlled by transfusion of a 4 per cent sodium bicarbonate solution. Mannitol gives good diuretic effect. Hyperkalaemia, which usually attends acute renal failure, is controlled by intravenous administration of 60-100 ml of a 20 per cent glucose solution with insulin (1 unit per 3 g glucose). A permanent catheter should be used to control the efficacy of treatment of renal failure (by measuring hourly diuresis). If diuresis decreases or the therapeutic effect is insignificant, haemoperfusion is indicated.

Nitrous oxide with oxygen can be given by mask during primary treatment of the burned areas. This ensures analgesia and is also a resuscitation measure in the general complex of antishock therapy. Atropine, antihistaminic preparations, promedol, and corticosteroids should be used for premedication. Shallow halothane-oxygen anaesthesia can also be effectively used.

If injuries are multiple and combined and the patient needs vast surgical correction, endotracheal anaesthesia should be used (with relaxation of muscles and controlled respiration).

## Toxico-septic Shock

Septic shock is characterized by acute cardiovascular insufficiency caused by intoxication due to the septic process. This shock arises in sepsis, peritonitis, vast phlegmona, pneumonia, and pleural empyema.

*Clinical picture* This is characterized by a sudden deterioration of the general condition, first high and then subnormal body temperature, fall of arterial pressure, and a marked impairment of micro-

circulation (the skin is cold, pallid and cyanotic) Consciousness is dimmed (up to coma) Symptoms of renal failure (oliguria and anuria) increase

The specific *therapy* includes intravenous administration of big doses of broad-spectrum antibiotics, hyperimmune preparations, and big doses of corticosteroids Haemoperfusion should be conducted during stabilization of blood circulation (replenishment of the circulating volume, elimination of microcirculatory disorders) and during blood transfusion Oxygen therapy should be conducted continuously Hyperbaric oxygenation should be conducted, if possible

### Anaphylactic Shock

This is a severe allergic reaction similar in its symptoms to acute cardiovascular and adrienal failure The therapy is substantially the same as described above, except that corticosteroids, antihistaminics, adrenaline, and its derivatives should be administered repeatedly Oedema should be controlled by dehydration therapy

## Chapter 20

### Intensive Therapy of Toxaemia

Toxaemia (toxicosis) is a severe non-specific reaction of a child to toxins or viruses Toxaemia occurs mostly in sensitized children with hyperergic reaction, but it can also occur in children with flaccid reaction In both cases the neurohumoral and endocrine regulation and the main types of metabolism are upset Toxaemia can be secondary to many diseases or disease conditions, e g acute surgical diseases, pneumonia, acute respiratory diseases, intestinal infections, and the like Alimentary toxaemia with exaemia and neurotoxia is usually regarded as an independent nosological form

#### Alimentary Toxaemia with Exaemia

Alimentary toxaemia with exaemia usually occurs in nurslings and infants with intestinal infections caused by shigellae, yersiniae, Coli bacteria, salmonellae, and staphylococci

*Pathogenesis* The disease is caused by endotoxins released from destroyed gram-negative bacteria The toxins react with the formed elements of blood to give compounds with high sympathomimetic activity As a result, the vessels are constricted to impair peripheral circulation and cell metabolism The increased capillary permeability in the gastrointestinal mucosa and the changed venous pres-

sure increase the average hydrostatic capillary pressure and displace large volumes of liquid from the intravascular space into the extravascular space. The patient develops diarrhoea (with loss of liquid and salts) and acidosis, the central blood circulation is disordered. The upset water-salt balance and acidosis concur with decreased tone of the peripheral arterial sphincters, while the tone of the post-capillary sphincters remains normal. This increases blood reserves and decreases the venous backflow. The central venous pressure falls and the vascular permeability increases significantly. The compensatory passage of water and salts from the intra- and extracellular space into the vessels, and the loss of water and salts with vomit and diarrhoea, cause general dehydration of the body, which is the main symptom of alimentary toxaemia with exaemia.

*Clinical picture* Three degrees of alimentary toxaemia are distinguished, viz, mild, moderate and severe. Children with moderate and severe forms of toxaemia should be given intensive therapy. Depending on the character of disorders in the water-salt metabolism, three types of dehydration are differentiated in alimentary toxaemia. Their knowledge is important for administering correct therapy (see Table 22).

*Treatment* After the degree and type of dehydration in alimentary toxaemia have been established, it is necessary to determine the lacking volumes of liquid, electrolytes, protein, the rate of intravenous administration, and then to prescribe adequate anti-bacterial therapy, to restore diuresis, and to conduct detoxication measures. All these procedures should be done immediately and in succession. If vomiting is recurrent, the stomach should be irrigated with isotonic sodium chloride solution through a gastric tube, 30-50 ml of the solution should be left in the stomach and antibiotic added as a measure of actiotropic therapy. Aminazine can be added to decrease the vomiting reflex. The child should then be given only water and tea during 6-12 hours. This is the first phase of infusion therapy. A salt-glucose mixture or tea should be given to drink (depending on the type of dehydration). Intravenous infusions should then be conducted. A peripheral vein of the head, or the cubital vein, should be punctured or, if necessary, the central vein (subclavian or internal jugular) should be catheterized.

The daily volume of liquid to be administered should be determined by summing up the following: (a) the daily liquid demand of a healthy child, (b) the present liquid deficit in the range from 5 to 15 per cent, (c) continuing loss of liquid, which should be determined by weighing the cloths soiled with excretions and vomit.

The first problem of infusion therapy is to replenish the lost liquid in order to normalize central blood circulation. If the arterial pressure is normal, rheopolyglucin (or a 10 per cent glucose solu-



Table 22 Dehydration in Children with Alimentary Toxaemia

| Type of dehydration                          | Onset conditions   | Clinical picture   | Weight loss          | Laboratory findings  |
|--|--|--|----------------------|--|
| I Isotonic                                   | Moderate and severe infections, early stage of severe disease                            | Flaccidity, anxiety, Pallor, skin is dry Vomiting 2-3 times a day, stools 5-6 times a day, diuresis slightly decreased   | About 5 per cent     | Electrolyte content normal, compensated acidosis, haematocrit—maximum normal   |
| II Hypertonic (water-deficit, intracellular) | Frequent stools and vomiting, hyperpyrexia and dyspnoea                                  | Strong thirst, anxiety, eyes retracted, soft, oliguria, dry mucosa, fontanelle is not depressed Tachycardia Clonic-tonic convulsions are possible  | About 10 per cent    | High electrolyte content, metabolic acidosis, increased osmolality   |
| III Hypotonic (salt-deficit, extracellular)  | Develops gradually with vomiting prevailing over stools, at latter stages of the disease | Flaccidity, adynamia, marble skin, refusal of drinks, the fontanelle sinks, cardiovascular failure, general hypotension, the skin fold levels slowly Tonic convulsions are possible Oliguria | About 10-15 per cent | Low electrolyte content and osmolality, decreased circulating blood volume, metabolic acidosis Haematocrit, total protein and urea are increased |

tion) should first be transfused. In the presence of hypotension 5-10 per cent albumin, polyglucin, or plasma should first be administered. The transfusion rate is sufficiently high 20-30 drops per minute. The drip should be continued until the central venous pressure in the child begins rising.

The second phase is substitution. Liquid deficit is eliminated and diuresis is forced with saluretics (to ensure detoxication and elimination of acidosis) within 24-48 hours. Electrolyte disorders should be corrected by adding electrolytes to glucose solutions in amounts necessary to meet the daily demands for them and to replenish their deficit, which is calculated from the laboratory findings and is determined during the process of its correction. It should be remembered that most blood substitutes contain sodium and there is a danger of its overdosage (especially in upset diuresis). Then the volume of enteral nutrients should be increased gradually, while the volume of transfused liquids decreased.

Aetiotropic treatment should be conducted enterally with antibiotics with proper consideration of the epidemiological situation and the clinical picture. Gentamycin, rifampicin, amikacin, and polymyxin should be prescribed. Some infections, e.g. colienteritis, salmonellosis, especially in infants to 6 months of age, run a course similar to that of generalized infections and require parenteral administration of antibiotics.

Moreover, during treatment of alimentary toxaemia use should be made of preparations improving peripheral circulation of blood and metabolism: complamin, trental, ganglioblockers, cocarboxylase, ATP, panangin, and vitamins.

### Neurotoxia

Neurotoxia can be secondary to many infections. This syndrome should be suspected in the presence of symptoms of affected central nervous system, excitation followed by depression, convulsions, hyperpyrexia, and meningeal symptoms.

*Aetiology and pathogenesis* Neurotoxia is caused by irritation of the central nervous system and its vegetative portions by bacterial toxins, viruses, and products of tissue decomposition. This causes reflex spasms of vessels with their subsequent dilatation. This is followed by local tissue hypoxia, increased vascular and cell membrane permeability. Proteolysis in cells releases osmotically active substances, which, in turn, increases cell hyperhydration.

Neurotoxia in infants is usually provoked by the following factors: inadequate regulation of metabolic processes, hydrolability, and the presence of liquid in amounts considerably exceeding those in older children.

*Clinical picture* Neurotoxia usually begins acutely with general anxiety, which is followed by dimmed consciousness and inhibition. As a rule, these symptoms develop in the presence of hyperpyrexia. Meningeal symptoms develop quickly, then comes tachypnoea and marked cardiac dysfunction. ECG shows displacement of the *T* interval and shortening the interval between the *T* and *P* waves. The arterial pressure usually increases and tachycardia intensifies. If this process is not arrested, oedema of the brain and lungs may occur. Oliguria and anuria are also characteristic.

*Treatment* Complex therapy of neurotoxia should first of all be directed at eliminating disorders in cell metabolism, oedema of the brain, hyperpyrexia, respiratory distress and heart failure.

1 Inadequate reflex reactions of the central nervous system can to a considerable measure be removed by the neuroplegics. Aminazine in combination with pipolphen is administered 0.1 ml of a 2.5% solution per year of age. In addition to these preparations, lytic mixtures also include antihistaminics (dimedrol and suprastin).

in various combinations Luminal (phenobarbital) may also be added in a dose of 2-3 mg/kg a day

2 Dehydration therapy is indicated for neurotoxicity. Diuretics are widely used (a) a very prompt effect is attained with furosemide (lasix) given in a dose of 3-5 mg/kg a day. Lasix remains in the circulating blood for 4 hours and repeated administrations of the preparation are therefore necessary. Best results are attained with the primary dose not less than 10 mg (1 ml). Repeated doses can be given per os, (b) if lasix is not available, mercury preparations, e.g. novurit, fonurit, should be used as diuretics (0.1 ml per year of age), (c) osmotic diuretics, e.g. mannitol (5 ml/kg of a 5 per cent solution, intravenously) and urea (5-10 ml of a 30 per cent solution, intravenous drip) should be better used for renal failure. Urea should be administered with strict control of diuresis and of urea concentration in the blood. It is recommended to abstain from urea in severe renal failure. A 20-30 per cent mannitol solution can be used instead.

3 Glycerol (0.5-2 g/kg a day) should be administered into the stomach through a gastric tube for dehydration.

4 A 10 per cent calcium chloride solution should be used to decrease vascular permeability and to increase diuresis. Or a 10 per cent calcium gluconate solution can be used in a dose of 1 ml per year of age.

5 A similar effect (together with a hypotensive and sedative action) is attained with a 25 per cent magnesium sulphate solution (1 ml per year of age). The solution should be injected deep into the muscle. A 5 per cent solution should be used for intravenous injections with isotonic sodium chloride solution as a diluent.

6 The therapy directed to arrest convulsions should be conducted, if necessary (see Ch. 24).

7 In the presence of hyperpyrexia, measures should be taken to decrease the temperature (see Ch. 23).

8 If the taken measures fail, 5-10 ml of cerebrospinal liquor should be withdrawn (the amount depending on the condition of the child and other conditions).

9 In the presence of heart failure, cardiac glycosides should be used. 0.05-0.1 ml of a 0.05 per cent strophanthin solution, 1-2 times a day, 0.1-0.2 ml of a 0.06 per cent corglycon solution, 1-2 times a day. These preparations should be administered in 10-15 ml of a 20 per cent glucose solution. Cocarboxylase (50-100 mg) should be administered simultaneously.

10 Collapse should be treated by taking rehydration measures. Plasma, glucose, polyglucin, and Ringer's solutions should be used for the purpose. Drip administration of the fluids in neurotoxicity is indicated only for collapse. Infusion of considerable amounts of liquid is otherwise contraindicated because they will promote oedema and swelling of the brain. Stimulating substances are indicated.

adrenaline and noradrenaline (1 1000) and 1 per cent mesaton solution, 0.1 ml per year of age

11 The hormonal preparations, prednisolone (1-2 mg/kg a day) and hydrocortisone (3-5 mg/kg a day) should be used

12 If neurotoxicity is moderate (without marked signs of brain oedema), the therapy should be directed at eliminating toxic substances from the body. Rehydration with simultaneous dehydration should be carried out for the purpose. Hypertonic solutions of glucose and insulin and isotonic sodium chloride solution are administered. The amount of liquid to be administered is 60-80 ml/kg a day. Administration of liquid should be conducted under strict control of diuresis.

13 Broad-spectrum antibiotics and semisynthetic antibiotics should be used to control intoxication.

14 Vitamin complexes and rutin are indicated. Oxygen therapy is obligatory.

## Chapter 21

### Intensive Therapy of Coma

Coma is a grave condition during which consciousness is either severely disturbed or absent, the reflexes are upset and the vital organs and systems dysfunction. Coma occurs due to profound depression of the cerebral cortex, which diffuses onto the subcortical and dependent parts of the central nervous system.

*Aetiology and pathogenesis* Coma can be caused by both endo- and exogenic factors. Endogenic factors are metabolic disorders (hypokalaemia, hypochloraemia), cerebral or liquor circulatory disorders, and tumours of the brain. This group also includes diseases of the liver and kidneys, diabetes mellitus, adrenal diseases, etc. Exogenic factors include infectious diseases, such as influenza, scarlet fever, dysentery, botulism, and the like conditions, various industrial and medicamentous poisonings, injuries, etc.

*Clinical picture* Two stages in the clinical picture of coma can be distinguished. They differ in the degree of inhibition of the central nervous system and disorders in the function of the vital organs. Stage I is precomatose (the first degree is mild, the second marked or of medium gravity) and stage II coma (the third degree, severe, and the fourth degree, terminal, i.e. extremely critical).

The first degree coma is characterized by dimmed consciousness, indifference, flaccidity of the child, who answers with difficulty, his speech is blurred. The child sleeps but wakes from extraneous stimuli. Respiration is slightly accelerated, the cardiac activity is satisfactory. Tachycardia is only insignificant. Arterial pressure is normal or slightly elevated. The reflexes are active.

Coma of the second degree is characterized by sopor (profound sleep), from which the child recovers with difficulty. When the child wakes up, he answers questions slowly. His speech is scanning and slurring. Respiration and the heart rate are accelerated. Arterial pressure is either normal or slightly decreased. The reflexes are weakened.

The third degree coma is characterized by loss of consciousness, the child does not wake up. All unconditioned reflexes are absent: corneal, pupillary, tendon, or swallowing. The pupils are dilated. The skin is greyish. Acrocyanosis. Respiration is fast, shallow and irregular. Arterial pressure is very low. The child defaecates and urinates involuntarily.

The fourth degree coma is also characterized by the absence of consciousness. Reflexes are absent, the child is motionless. Kussmaul or Cheyne-Stokes' respiration. Marked bradycardia with subsequent cardiac arrest. Arterial pressure is undeterminable. The terminal state converts into agony and clinical death. Spontaneous respiration is absent and the heart does not beat. The life can be maintained only by artificial respiration and cardiac massage.

This is the general clinical picture of coma. Depending on the cause, various forms of coma are distinguished. Each form is characterized by its specific symptoms, diagnosis and therapy.

When a child in coma is admitted to a hospital, it is difficult to identify the cause or type of coma. It is therefore necessary, if possible, to collect anamnesis, to clarify the circumstances under which coma developed and what diseases preceded it (e.g. renal diseases, diabetes mellitus, epilepsy). Child's complaints in the precomatose state are also important diagnostically. It is very important to examine the patient's belongings which may give some diagnostic hints (medicines, medical documentation). The child should be undressed completely in order to avoid diagnostic errors. The colour of the skin and mucosa should be inspected. Then the reaction of the pupils to light should be tested (in both eyes), the eyeballs should be felt to determine their tone, next determined should be the character and type of respiration and the smell of breath. The borders of the heart, liver and the spleen should be outlined. The arterial pressure should be measured and heart sounds auscultated. Pathological reflexes should next be revealed and physiological reflexes (knee jerk, Achilles tendon, abdominal) checked.

Laboratory tests should be carried out for blood sugar, acetone, urea, rest nitrogen, bilirubin, chlorine, potassium, sodium, and calcium. An unconscious child requires special care. If the diagnosis is unknown, it is first of all necessary to ensure and maintain the vital functions, and only then begin searching for the cause of unconsciousness.

## Diabetic Coma

*Aetiology and pathogenesis* Diabetic coma in children is caused by rapidly progressing metabolic disorders in diabetes mellitus with late diagnosis (undiagnosed diabetes mellitus) The factors provoking severe metabolic disorders and the onset of diabetic coma are inadequate diet (eating unrestricted amount of sweet and fatty food), suspension of insulin or other preparations decreasing the blood sugar, considerable reduction of their doses, operative interventions, physical or psychic trauma, infectious diseases, recurrent vomiting, diarrhoea, or prolonged hunger

The mechanism of diabetic coma is absolute and relative insulin insufficiency In the absence of sufficient insulin, utilization of glucose by tissues is upset, permeability of cell membranes for glucose decreases, and the synthesis of glycogen (mainly in the liver) is disordered, while its decomposition intensifies Glucose is formed from proteins and fats, the release of STH, ACTH, catecholamines, and glucagon (the antagonist to insulin) increases Disturbances in glucose absorption, formation and splitting of glycogen decrease its content in the body and increase sharply the glucose concentration in the blood (hyperglycaemia) The blood sugar increases to 16.7-27.8 mmole/l (300-500 mg%) against the normal 4.4-6.65 mmole/l (80-120 mg%)

Intensified lipolysis increases the release of free fatty acids into the blood Free fatty acids are underoxidized and the production of ketone bodies (acetoacetic and beta-oxbutyric acids, acetone and cholesterol) in the liver increases Proteins decompose increasing the amount of amino acids, rest nitrogen and ammonia in the blood Lactic acid is formed in the muscles from glycogen in large amounts All this stimulates the onset of metabolic acidosis Insulin deficit and metabolic acidosis promote the release of potassium and other electrolytes from cells

Hyperglycaemia, hyperketonaemia and hyperazotaemia promote excretion of glucose, ketone bodies and rest nitrogen with the urine Renal reabsorption decreases and the diuresis increases Sodium, potassium, phosphorus, and chlorine are withdrawn from the body with the urine in great amounts Loss of sodium with urine decreases the capacity of tissues to hold water, which intensifies polyuria Pathological loss of liquid with Kussmaul's respiration and with vomitus and diarrhoea aggravates dehydration Hypovolaemia develops, which causes severe circulatory disorders arterial pressure falls, stroke volume diminishes, and filtration decreases Collapse develops and diuresis diminishes to anuria

The mechanism of affection of the central nervous system in diabetic coma has a complicated genesis Hyperketonaemia, acidosis and hypoxia of the brain are the leading factors

The most typical clinico-metabolic version of diabetic coma in children is ketoacidotic coma (78 per cent) Hyperosmolar coma occurs in 8 per cent and hyperlactataemic coma in 14 per cent of cases Hyperosmolar and hyperlactataemic coma occur mostly in the aged

*Clinical picture* Diabetic coma is often preceded by a period of precursors, such as fatigue, thirst, loss of appetite, sleepiness, apathy, nausea, vomiting, and headache. During this period, in addition to hyperglycaemia, the blood analysis shows also decreased alkali reserve, ketosis and acidosis, acetone is found in the urine If not treated properly, the clinical picture of diabetic coma develops The patient is unconscious, the face is pallid or hyperaemic, cyanosis is absent The skin and mucosa are very dry and cold Acetone breath can be felt at a distance Turgor is decreased The eyeballs are soft Respiration is slow and pathological (Kussmaul's respiration) The pulse is fast, weak and small Arterial pressure is low, the heart sounds are weak The tendon reflexes are decreased or absent The pupils are moderately wide Vomiting occurs Vomitus sometimes looks like coffee grounds The liver is usually enlarged Pain in the abdomen is sometimes so severe that the picture of acute abdomen is often simulated This can mislead the physician and a surgical operation is often unreasonably performed to impair the patient's condition The body temperature is usually normal or subfebrile

Laboratory tests for sugar in the blood and urine show hyperglycaemia which, in severe cases, can be as high as 27.8-55.5 mmole/l (500-1000 mg%) Glucosuria may be 50-100 g/l (5-10 per cent) The urine contains sugar and acetone bodies The specific gravity of the urine is high Blood analysis shows acidoketosis and azotaemia. Leucocytosis is characterized by the shift to the left.

A child with diabetic coma may have decreased renal function and the sugar of urine can thus decrease considerably In some cases it is absent altogether

*Diagnosis* A certain sequence of symptoms is characteristic of hyperglycaemic coma Gradual loss of consciousness, acetone breath, respiratory distress, cardiac dysfunction, hypotonia of the eyeballs, supplemented by laboratory findings, help diagnosing diabetic coma It is necessary to remember that acetone can be found in the urine of children during post-operative period due to the development of secondary toxemia Testing blood for alkaline reserve is an accurate differential-diagnostic tool  $\text{CO}_2$  level above 40 per cent indicates secondary toxemia

*Treatment* This should be a complex therapy including insulin and control of acidosis and dehydration

1 Insulin therapy includes the following (a) administration of insulin in strictly individual doses To this end it is necessary to find out if the patient was earlier treated with insulin and what were

the doses, the time of administration of the last dose, it is important to know if the patient was given prolonged-action insulin (protamine zinc insulin) When calculating the insulin dose, it is necessary to take into account its last dose,

(b) if the child was never treated with insulin before, it should be given in a dose of 1U per kg body weight Infants under 7 years of age should be injected 15-20 units, while older children 20-30 units,

(c) if a child was earlier treated with insulin, a full daily dose (which was earlier given to the child before the onset of coma) should be injected In order to prevent hypoglycaemia, it is necessary to administer a 5 per cent glucose solution (intravenous drip) diluted with an equal volume of an isotonic sodium chloride solution,

(d) repeated injections of insulin should be done at 1-2-hour intervals (within the first 6 hours) As the condition improves, the interval between subsequent injections can be increased,

(e) ordinary insulin should be injected during the first days after recovery from coma The preparation is administered 3-4 times a day with testing blood and urine for sugar Later insulin is administered twice a day, usually before breakfast and before tea in the afternoon,

(f) the overall insulin dose to recover the child from coma should not exceed 150 units, but in some rare cases the dose may considerably exceed this maximum

2 Acidosis is controlled by administration of a 4 per cent sodium bicarbonate solution

3 Isotonic sodium chloride solution (8-10 ml/kg) should be injected intravenously (simultaneously with the first insulin dose) Then follows infusion by drip The amount of liquid to be infused is 100-150 ml/kg During the first 6 hours, the fluid consisting of  $\frac{1}{3}$  isotonic sodium chloride solution,  $\frac{1}{3}$  Ringer's solution, and  $\frac{1}{3}$  5 per cent glucose solution should be administered Fluid for further infusion contains more glucose and potassium Not less than 1 g of glucose should be contained per each unit of insulin When planning infusion therapy, the amount of fluid to be injected should be calculated with reference to the normal requirement plus pathological loss of liquid

4 If a child regains consciousness, he should be given half-sweet tea, fruit juice, and alkaline mineral water (Borzhomi)

5 To prevent secondary infection during diabetic coma, antibiotics should be administered Vitamins B<sub>1</sub>, B<sub>2</sub> and C should be added in large doses to the dropper with isotonic sodium chloride solution

6 Complex therapy should obligatory include lipotropic substances, e.g. methionine, lipocaine, or curds

7 The diet should then be extended gradually to a physiological one with a certain fat restriction



## Hypoglycaemic Coma

*Aetiology and pathogenesis* Hypoglycaemic coma is caused by insulin overdosage (during therapy of diabetes mellitus or due to insufficient carbohydrate intake with food) Decreased absorption of glucose in the brain cells is the major pathogenetic factor of hypoglycaemic coma The cerebral cortex dysfunction occurs with oedema and necrosis of some portions of the brain Hypoglycaemia irritates the hypothalamus and the adrenal medulla The release of adrenaline increases, the function of ACTH (glucocorticoid system and STH production) intensifies This is the compensatory mechanism directed at normalization of blood sugar

*Clinical picture* The patient feels very hungry and is able to eat much food The child is irritable, frightened, he complains of double seeing, his motor function is upset Sometimes the child is anxious,

Table 23 Differential Diagnosis of Diabetic and Hypoglycaemic Coma (according to Bubnova and Martynova, 1963)

| Diabetic coma  | Hypoglycaemic coma                            |
|--|---|
| Predisposing factors                                   |   |
| 1 Insufficient insulin dose                            | 1 Insulin overdosage                          |
| 2 Inadequate regimen (fat abuse)                       | 2 Insufficient nutrition after insulin        |
| Development of coma                                    |   |
| 1 Prodromal phase                                      | 1 Rapid and sudden                            |
| 2 Gradual loss of consciousness                        | 2 Rapid loss of consciousness                 |
| Symptoms   |   |
| 1 Dry skin Dehydration Cyanosis of the skin and mucosa | 1 Pallor and hyperhidrosis                    |
| 2 Dry tongue   | 2 Moist tongue                                |
| 3 Muscular hypotonia                                   | 3 Muscular rigidity, trismus of jaws          |
| 4 No convulsions                                       | 4 Convulsions Babinski's syndrome             |
| 5 Hypotonia of eyeballs                                | 5 Normal tone of the eyeballs                 |
| 6 Kussmaul's respiration                               | 6 Normal respiration                          |
| 7 Fast and small pulse                                 | 7 Bradycardia is possible                     |
| 8 Appetite is absent, nausea and vomiting              | 8 Increased appetite                          |
| 9 Abdominal syndrome in some cases                     | 9 Abdominal syndrome is absent                |
| 10 Temperature subnormal                               | 10 Temperature usually normal                 |
| 11 Hepatorenal syndrome is frequent                    | 11 Peripheral blood normal                    |
| 12 Acetone breath                                      | 12 Acetone breath is absent                   |
| 13 Acetonuria and glucosuria                           | 13 Acetonuria or glucosuria is usually absent |
| 14 Hyperglycaemia                                      | 14 Hypoglycaemia or normal blood sugar        |
| 15 Decreased alkaline reserve                          | 15 Normal alkaline reserve                    |
| 16 Hyperketonaemia                                     | 16 Hyperketonaemia is absent                  |

he talks loudly, laughs, makes faces, hyperkinesis, hallucinations, tonic and clonic convulsions develop. As distinct from diabetic coma, hypoglycaemic coma occurs suddenly. The child loses consciousness within minutes. Hypoglycaemia is characterized by profuse sweating, moist tongue, shallow and rhythmical respiration. Hypotonia of the eyeballs is absent; no acetone breath. The skin is moist and pallid. Trismus of the jaws develops. Uni- or bilateral Babinski's sign occurs. If trismus of the jaws and Babinski's sign occur in a child during treatment of diabetic coma, insulin administration should be suspended and treatment with glucose solutions begun. Heart sounds are dull during coma. Arterial pressure is labile. The pulse is irregular and fast. The blood contains increased amounts of leucocytes and lymphocytes, leucopenia is sometimes observed, the sugar level is low. Sugar or acetone are absent from the urine.

Differential diagnostic signs of diabetic and hypoglycaemic coma are given in Table 23.

The *treatment* of hypoglycaemic coma should be started immediately. If coma continues for more than 3 hours, severe pathological changes are likely to occur in nerve cells with subsequent changes in the central nervous system. Concentrated glucose solutions (preferably 20 or 40 per cent solutions, without insulin) should be administered. Glucose should be infused until signs of consciousness reappear. When the child is conscious, he should be given sweet tea, jam, honey and other sweets, and wheat bread. The sugar level in the blood should be controlled. Therapy of hypoglycaemic coma should obligatorily include oxygen and hormone therapy.

### Azotaemic Coma (Uraemia)

*Aetiology and pathogenesis* Uraemia is the terminal stage of renal failure. It occurs due to renal dysfunction in acute or chronic diseases. Uraemia can be caused by glomerulonephritis, pyelonephritis, amyloid nephrosis, and some other diseases.

Excessive amounts of nitrous products are retained in the blood and tissues and great changes occur in the acid-base balance along with acidosis. Intoxication is another very important factor in pathogenesis of uraemia.

*Clinical picture* Comatose condition develops gradually and is usually preceded by precursors: the child excretes ample light urine with low specific gravity (mostly during night time). The central nervous system becomes inhibited gradually: weakness, headache, sleepiness, apathy, irritability, then thirst, dry mouth, and skin itching develop. The skin is dry with traces of scratching. The appetite is poor. The mouth is affected with ulcerative stomatitis. Nausea, vomiting, and sometimes diarrhoea develop. The vomitus looks like coffee grounds or has streaks of blood. Diuresis gradually decreases,

vision becomes impaired, the body temperature drops. Anaemia is marked. The skin is affected with eruption and small haemorrhages. As coma develops, the child's consciousness becomes dimmed, he is restless, tosses in his bed. Muscular twitching converts to clonic convulsions. Epileptiform fits are possible. The breath smells of urea. Urea crystals can sometimes be seen on the skin. The mucosa has signs of necrotization. The nose and gums are bleeding, the injection sites are marked by haemorrhages. The pulse is superficial, irregular and fast. Systolic murmur can be heard at the heart apex, pericardial and pleural friction can also be heard. Examination of the eye bottom reveals oedema of the optic nerve papillae, contraction of the arteries, and dilatation of the veins. Meningeal symptoms develop: hemiparesis, aphasia, miosis, derangement of vision. The tendon reflexes are increased, Babinski's sign is observed. The urine is light and with low specific gravity (1 000-1 007), it contains protein, red blood cells and casts. Rest nitrogen of blood increases significantly 142.8 mmole/l (over 200 mg%), the urea content is also very high. The erythrocyte count decreases significantly. As these symptoms further progress, the child develops pathological Kussmaul's or Cheyne-Stokes respiration.

*Diagnosis* The condition should be differentiated from diabetic coma. Table 24 gives differential diagnostic signs of these diseases.

Table 24 Differential Diagnosis of Azotaemic and Diabetic Coma

| Symptom                             | Diabetic coma      | Azotaemic coma                        |
|-------------------------------------|--------------------|---------------------------------------|
| Blood sugar                         | Increased          | Normal                                |
| Pulse                               | Soft and fast      | Tense and firm                        |
| Eyeballs                            | Decreased tone     | Normal tone, exophthalmos is possible |
| Respiration                         | Slow and deep      | Pathological                          |
| Fundus oculi                        | Diabetic retinitis | Albuminuric retinitis                 |
| Tendon reflex                       | Absent             | Increased                             |
| Breath                              | Acetone breath     | Urea breath                           |
| Urine acetone                       | Present            | Absent                                |
| Test with anhydrous ferric chloride | Positive           | Negative                              |
| Specific gravity of urine           | High               | Low                                   |

*Treatment* This should be a complex therapy aimed, in the first instance, at elimination of the comatose state and treatment of the main disease.

1 The diet in uraemia should be rich in fats and carbohydrates. If the child is unable to eat, nutrition should be given through a gastric tube. Olive oil and eggs in a 20 per cent glucose solution is an effective mixture. In the absence of oedema the child can be given to

drink moderate amounts of liquid Sodium chloride (table salt) should be restricted to prevent oedema The amount of protein ingested should be decreased in the precomatose condition

2 Detoxication therapy includes (a) 5 per cent or 10 per cent glucose solution, by intravenous drip; (b) hypertonic plasma solution, (c) plasma substitutes, (d) neocompensan, which is an effective detoxicating preparation, (e) mannitol (osmotic diuretic) increasing diuresis and decreasing urea concentration in the blood, (f) in the presence of hyperkalaemia glucose solutions with insulin and a 10 per cent calcium chloride solution should be administered intravenously

3 Big doses of vitamins C and B, and also of cocarboxylase should be added to solutions for intravenous drip

4 Emulsified fats should also be administered intravenously

5 In the presence of excitation or convulsions chloral hydrate should be administered by enema, sodium oxybate is also given Symptomatic therapy should be conducted for cardiovascular pathologies

6 Camphor spirit or toilet vinegar can be used to remove skin itching Diathermia can be applied to the region of the kidneys Paranephric procaine block is also effective

7 The removal of rest nitrogen from the body can be improved by (a) blood-letting; (b) replacement blood transfusion, only fresh blood can be transfused (not later than in 2-3 days after its taking), (c) gastric dialysis with a weak alkali solution (through a gastric tube), (d) intestinal dialysis with a 10 per cent sodium chloride solution (through siphon enema), (e) haemodialysis with an 'artificial kidney' apparatus, (f) peritoneal dialysis is less effective than haemodialysis, but the gradual clearance procedure is better tolerated by the child than haemodialysis Efficacy of peritoneal dialysis may be improved if the time of perfusion of the dialytic solution is increased.

### Eclamptic Coma

*Aetiology and pathogenesis* It usually occurs in women during their second half of pregnancy, but can also occur in children with acute diffuse nephritis Pathogenesis of eclampsia is based on increased intracranial pressure caused by oedema and swelling of the brain and upset cerebral circulation, which causes hypoxia of the brain

*Clinical picture* Coma precursors are numbness, anxiety, excruciating headache, oedema, and hypertension The onset is sudden the child takes a deep breath, emits a cry loses his consciousness, develops tonic convulsions involving the respiratory muscles and the diaphragm, clonic convulsions of the entire body then follow The face is markedly cyanotic, acrocyanosis and trismus are observed, foaming saliva appears at the mouth Arterial pressure is high, the pulse

slow and tense, respiration is slow, irregular and gurgling. The attack lasts for 3 to 20 minutes; it is characterized by mydriasis, increased tendon reflexes, pathological reflexes, and strabismus, the pupils do not react to light. The urea and creatinine of blood are normal or slightly increased. The urine contains protein, red blood cells and leucocytes. Convulsions may recur at 30-60-minute intervals. Focal symptoms (hemiparesis, hemianopia, aphasia), and punctate haemorrhages into the retina sometimes occur after the attacks.

*Diagnosis* It is necessary to differentiate eclampsia from epileptic or azotaemic coma. Epileptic coma is characterized by a more energetic onset and the anamnesis has no indications of preceding nephritis; oedema, bradycardia or hypertension are absent. Azotaemic coma begins gradually, the early comatose condition is less profound, the specific gravity of the urine is low, and the smell of urea can be felt at a distance.

*Treatment* 1 The therapy of eclampsia begins with blood-letting.

2 Cold should be applied to the head.

3 Chloral hydrate enema should be given in the intervals between convulsions.

4 Sodium oxybate or a 2 per cent hexenal solution is administered during attacks until convulsions discontinue.

5 Hypertonic solutions of glucose (for dehydration), magnesium sulphate and calcium chloride should be used intravenously. Diuretics should also be given.

6 Lumbar puncture is indicated in the presence of prolonged convulsions. As a rule, the liquor is forcibly ejected.

7 After termination of convulsions luminal and aminazine are administered. Short-wave diathermy should be applied to the region of the kidneys. Paranephric novocaine block is effective.

### Post-traumatic Coma

*Aetiology and pathogenesis* This coma occurs after severe injuries, especially of the head. Coma is explained by increased intracranial pressure due to haemorrhage in the cerebral cortex, oedema of the pia mater, and increased liquor pressure in the cerebral ventricles. The severest coma occurs in open injuries of the skull and fractures of its base.

*Clinical picture* The child is unconscious. Vomiting and bradycardia are characteristic. Respiration is slow, the skin is pallid. Bleeding from the mouth, nose and ears is frequent. The temperature elevates, the Kernig symptom is blurred. The tendon reflexes are decreased in the lower extremities, the pupils are dilated, their reaction to light is weak or absent. Nystagmus, anisocoria, difficult swallowing and respiration are observed. Leucocytosis is marked, with a shift to the left. Urine retention is common.

*Diagnosis* This is not difficult. The presence of haematoma or wounds indicates trauma. Additional methods of examination are roentgenography of the skull, echoencephalography and cerebrospinal puncture. The liquor is tarred with blood.

*Treatment* This should mainly be directed at stoppage of bleeding and elimination of haematoma (subdural, epidural). This is attained by surgical treatment of the wound or trepanation of the skull, if necessary. Among other measures are (a) haemostatic treatment with vikasol, haemophobin or calcium chloride (in age doses), (b) absolute rest; (c) cold on the head; (d) dehydration therapy (intravenous administration of hypertonic solutions of glucose, sodium chloride and diuretics, such as lasix, or mannitol), (e) decreasing vascular permeability by administration of a 10 per cent calcium chloride (intravenously) and 25 per cent magnesium sulphate solution (intramuscularly, 1 ml per year of age), (f) decreasing intracranial pressure by lumbar puncture (it should be done for both therapeutic and diagnostic purposes), 2 or 3 ml of the liquor should be released; (g) after the patient's condition improves, a surgical operation can be carried out. During subsequent days the patient should be given infusion therapy to meet the normal daily requirement plus the amount of liquid necessary to correct water-electrolyte disorders, because dehydration therapy causes marked disorders in all types of metabolism, (h) antibiotics should be administered in open injuries of the skull. Symptomatic therapy should be carried out in the presence of symptoms of any dysfunction.

### Hypochloraemic Coma

*Aetiology and pathogenesis* Hypochloraemic coma occurs due to decreased chloride content of blood in cases with incoercible vomiting, chronic nephritis or diarrhoea. Decreased chloride content upsets protein and mineral metabolism. Hypochloraemia occurs in stenosed pylorus, adrenal hyperfunction and Fanconi's syndrome.

*Clinical picture* The precomatose condition is characterized by frequently recurring incoercible vomiting and strong diuresis. When coma develops, vomiting is also incoercible, tetanic convulsions develop. Arterial pressure falls, tachycardia develops, the tendon reflexes are inhibited, and meningeal symptoms develop. The condition is characterized by dehydration, thickening of the blood (high haemoglobin and haematocrit), dry skin and mucosa. Azotaemia and hypocholesterolaemia occur. The body temperature decreases.

*Diagnosis* This is not difficult. Frequent incoercible vomiting, convulsions and laboratory findings (blood chlorine and other tests) are sufficient for establishing a correct diagnosis.

*Treatment* Normal chloride concentration in the blood and tissues should be restored. To that end, hypertonic or isotonic sodium chloride solution should be administered intravenously. Blood transfusion and symptomatic therapy should be conducted.

### Adrenal Coma

*Aetiology and pathogenesis* Adrenal coma develops gradually in the presence of chronic adrenal insufficiency (Addison's disease), or its onset is sudden in acute organic or functional affection of the adrenal glands. The provoking factors are strong emotional stress or psychic trauma. Adrenal coma occurs sometimes in neonates borne in asphyxia due to haemorrhage in the adrenal glands (after placing forceps). Coma can be caused by hyperplasia of the adrenal glands combined with defects of their growth, thrombosis of the adrenal vessels during sepsis, toxic affection during severe infectious diseases, haemorrhage in the adrenal glands in haemorrhagic diathesis, and diseases of the adrenal glands (tuberculosis, syphilis). Adrenal coma can also occur due to an abrupt suspension of hormone therapy in the post-operative period, insufficient amounts of administered hormone preparations, especially if the child was earlier treated with adrenal cortex hormones.

Pathogenesis of adrenal coma depends on the disordered water-electrolyte, carbohydrate and protein metabolism, increased capillary permeability, upset glucose oxidation in tissues, and excretion of large amounts of potassium and phosphates.

*Clinical picture* The most characteristic sign of adrenal coma is the vascular collapse. Marked pallor and cold sticky sweat suddenly appear. The child is excited, he complains of pain in the belly, but the excitement is very soon replaced by flaccidity, adynamia, and then unconsciousness. The lips and the nasolabial triangle become cyanotic, cyanosis spreads onto the other parts of the body. Dark spots and bright red petechial eruption appear on the skin of the back, arms and legs. Respiration becomes pathological (Cheyne-Stokes). The child lacks air. The pulse is fast and thready. Arterial pressure falls. The heart sounds are dull and arrhythmic. Dehydration and oliguria develop quickly. Rest nitrogen, leucocytes, eosinophils, and monocytes increase in blood. Hyponatraemia and hypochloraemia develop with increasing excretion of sodium and chlorine with the urine.

*Diagnosis* The diagnosis of adrenal insufficiency can easily be established provided the physician knows the previous condition of the child. It is sometimes necessary to differentiate adrenal coma from pneumomediastinum (especially after operations on the chest). Puncture of the mediastinum and chest x-ray provide evidence of air presence in the mediastinum. Anamnesis is important. It is necessa-

ry to know when hormone preparations have been suspended, what were the doses before and after the operation, before the therapy was suspended, etc

*Treatment* Treatment should be begun immediately to correct the deficit of adrenal cortex hormones. Hydrocortisone and prednisolone should be administered intravenously in a dose of 4-5 and 1-2 mg/kg, respectively. Later therapy should be directed at controlling collapse, dehydration, and correcting water-electrolyte metabolism.

1 Collapse should be controlled by intravenous drip administration of a 1 per cent mesaton and norepinephrine in isotonic sodium chloride solution. Isotonic sodium chloride solution, 5 per cent glucose solution, vitamins, and carboxylase should be administered (intravenous drip) for rehydration and normalization of the water-electrolyte balance.

2 After cessation of vomiting and regaining consciousness the child should be given per os a mixture of an isotonic sodium chloride and a 5 per cent glucose solution (1:1).

3 Since much potassium is lost, the hormone therapy should obligatory include potassium preparations, which should be given per os or intravenously with control of potassium excretion with the urine.

4 The rapid hormone therapy quickly eliminates adrenal coma. Further efforts of the physician should be directed at treatment of the main disease.

## Chapter 22

### Intensive Therapy of Brain Oedema

Oedema of the brain is its enlargement due to upset circulation of blood and water-salt metabolism, and some other factors.

*Aetiology and pathogenesis* Oedema of the brain can occur in many diseases involving the nervous system, e.g. influenza, pneumonia, toxæmia, peritonitis, trauma of the skull, and also in metabolic disorders (hypoproteinaemia) and ionic disbalance. Hypoventilation is one of the causes of brain oedema. It increases permeability of the vessels, disturbs the 'pump chest' mechanism, thus decreasing the blood flow to the right heart chambers. Hypercapnia can also provoke brain oedema. But the main cause of this phenomenon is hypoxia of various aetiology. Some authors believe that anoxic and ischaemic encephalopathies are attended by injuries of the vessels and necrosis of the parenchyma, especially of the cells with low oxidizing activity of the enzymes. According to other authors, hypoxia with normal elimination of  $\text{CO}_2$  does not cause brain oedema, which only occurs in cases where hypoxia combines with hypercapnia. Brain



oedema can thus be caused by diseases provoking hypoxia, hypercapnia, and water-electrolyte disbalance severe toxic pneumonia, acute respiratory diseases with grave respiratory dysfunction, toxicosis of various aetiology, and some other diseases.

Oedema is caused by penetration of fluid from the circulating blood into the brain. Oedema often combines with swelling of the brain. According to some authors, the pathogenesis of these conditions differs: brain oedema is caused by accumulation of fluids poor in proteins in the tissue fissures due to disordered permeability of the vessels, while in brain swelling water is bound in colloids due to their hydrophilic properties.

There is a vicious circle in pathogenesis of brain oedema: hypoxia, trauma, toxic invasion, and some other factors injure cells of the brain and the vascular walls to increase their permeability, thus causing oedema of the brain. The latter, in turn, injures (directly and indirectly) the cells to interfere with venous outflow, thus increasing intracranial pressure, inhibiting respiration, and causing other disturbances. These factors, in turn, increase oxygen deficit in the brain, and intensify hypoxia and metabolic disorders.

*Clinical picture* The picture of brain oedema presents certain difficulties for diagnosis. Neurological symptoms indicate brain oedema only in those cases where the main disease is known and these symptoms were absent earlier. Otherwise, neurological symptoms characterize the localization of the process but do not indicate the presence of brain oedema. The main symptoms of brain oedema are headache, vomiting and deranged consciousness, indicating intracranial hypertension. The degree of derangement can vary from a slight dimness to a deep coma. Consciousness can remain normal in chronic brain oedema, while in acute cases coma develops suddenly. Loss of consciousness is evidently connected not only with increased intracranial pressure but also with metabolic disorders in the brain and anatomical injuries. Consciousness can be deranged by disturbances in the cerebral circulation. Considerable retardation of the blood flow-rate causes sopor. A 50-60 per cent decrease in the circulation decreases oxygen consumption to cause hypoxia. The oxygen tension in the venous blood of 19 mm Hg is critical and causes loss of consciousness. In acute cerebral circulatory disorders, psychic disorders occur when only a small portion of the cells is affected by anoxia.

Among symptoms of cerebellar strangulation are irritation of the nervus vagus caused by its compression, hypoxia of medulla oblongata and its oedema in acute cases. Bradycardia, bradypnoea, sudden vomiting, dysphagia, paraesthesia of the shoulders and arms (less frequently in the region innervated by the trigeminal nerve) develop. A relatively common symptom is rigidity of the occipital muscles. It develops in the absence of the other symptoms of cerebel-

lar strangulation. The most severe symptom is arrest of respiration, which can occur quite unexpectedly without any precursors.

The clinical picture of brain oedema differs depending on duration and localization of the process, and on the gravity of the cerebrocranial injury. Sometimes, flaccidity, weakness, headache, sleepiness, and vomiting develop in the presence of the main disease. Paresis or paralysis occur or intensify, and congestion appears in the papillae of the fundus oculi. In other cases, as the oedema progresses and extends onto the stem region, convulsion attacks occur along with increasing flaccidity, sleepiness, cardiovascular and respiratory disorders, upset thermal regulation, and the development of pathological reflexes. The patient dies from dysfunction of the vital organs and systems.

The clinical theory of the main symptoms of brain oedema is based on the fact that the main factor, determining the progress of the affection, is the ratio between the arterial and intracranial pressure. Acute rise of the intracranial pressure stimulates elevation of the arterial pressure to the level slightly above the pressure of the cerebrospinal fluid. When the pressure over the surface of the medulla oblongata is equal to diastolic pressure or mean arterial pressure, hypoxia of the medullar centres develops. As a result, impulses are generated in the vasomotor centre, which pass the cervical part of the spinal cord to reach vasoconstrictors responsible for the elevation of the arterial pressure. Symptoms of excitation of medulla oblongata develop: elevation of arterial pressure, bradycardia, tachypnoea, and vomiting. When compression of the stem is especially severe and prolonged, the initial excitation of the centres is followed by their paralysis, which is attended with a fall in the arterial pressure, acceleration of the pulse, and respiratory arrest. This picture is observed in cases where the condition arises suddenly, and intracranial hypertension rapidly develops. If the process progresses slowly and the brain is able to compensate for the increasing pressure, these symptoms can be absent.

*Diagnosis.* Diagnosis presents certain difficulties. This especially holds for secondary oedema of the brain. The following measures are helpful in modern diagnosis of brain oedema.

(a) Skull x-ray. The picture shows changes in the region of sella turcica, retraction of the finger impressions, and separation of sutures indicating increased intracranial pressure. Disjunction of sutures is the first sign in infants.

(b) Electroencephalography. 'Pure' oedema of the brain is a rare clinical case. It is therefore difficult to interpret the EEG results, the more so that there is no unanimity in assessing EEG findings. Some authors maintain that brain oedema properly has no effect on EEG. In their opinion, the slow waves are generated by anatomic injuries surrounded by perifocal oedema. A more diffuse oedema is

accompanied with diffuse slow waves, which appear due to multiple necrotic foci in the brain tissue, or due to a severe cerebral anoxia. Other authors believe that EEG findings can be used to diagnose cerebral oedema.

(c) A very important diagnostic test is cerebrospinal puncture. The pressure, which is measured during the puncture, is proportional to the intracranial liquor pressure, provided the cranial and the lumbar subarachnoidal spaces are communicated. In the presence of block caused by displacement of the brain, the pressure of the cerebrospinal fluid can be normal or even slightly decreased, despite the elevated intracranial pressure. Liquor pressure above 130 mm H<sub>2</sub>O indicates the presence of cerebral oedema.

In addition to the mentioned tests, helpful also are pneumography of the brain, angiography and scanning with radioactive isotopes. The progressive neurological symptoms, and also agreement between the arterial and liquor pressure, are diagnostically important as well.

*Intensive therapy* The therapy of brain oedema should be directed at elimination of the main disease, which caused oedema. But it is only possible in cases where this disease is known. Moreover, one may never be sure that oedema can be removed by eliminating the cause (Plate 8).

1 Dehydration therapy is an important means of treatment of cerebral oedema. The therapy includes the following:

(a) Administration of hypertonic 10 per cent sodium chloride, calcium chloride solutions, and a 25 per cent magnesium sulphate solution. It is believed that hypertonic solutions of glucose can have a negative effect: first they decrease oedema considerably but 2-3 hours later the oedema progresses to become larger than before. Our experience shows that 10-20 per cent glucose solutions administered in small amounts together with other anti-oedema substances give an adequate clinical effect.

(b) Diuretics should be given to attain dehydration. Mercury diuretics (novurit, fonurit) should be given in a dose of 0.1 ml per year of age. Furosemide (lasix) is an effective preparation. It is given in a dose of 3-5 mg/kg a day. Lasix circulates in the blood during 4 hours. Good effect is attained if the first dose is not smaller than 10 mg.

(c) Osmotic diuretics. If the renal function is adequate, a 30 per cent solution of urea (1 g/kg) is administered. It has been established that urea denatures proteins, converting them from hydrophilic into hydrophobic substances, thus causing dehydration. But evidence has recently been obtained showing an irritating effect of urea on the venous walls, which can cause hyper- or hypovolaemia, and also the 'recoil' effect: in 6-12 hours after administration of urea, the intracranial pressure can rise again due to urea diffusion into the brain.

tissue where osmotic pressure thus increases. Repeated administrations of urea are therefore required at 6-12-hour intervals.

The best osmotic diuretic is mannitol. This is a preparation with a pronounced diuretic effect. It is not toxic, has no 'recoil' effect, nor does it pass into the cerebrospinal fluid, the preparation is effective with repeated administrations. A 10-30 per cent mannitol solution is used. A 30 per cent solution is more effective for brain oedema. The preparation is administered intravenously (rapidly) in a dose of 1 g/kg. Mannitol quickly decreases the intracranial pressure and easily dissolves in small amounts of water, it does not overload the cardiovascular system, nor does it cause necrosis of tissues at the site of injection. It is always ready for use as an aqueous solution. The preparation is indicated for renal dysfunction.

(d) Glycerol is now widely used per os in a dose of 0.5-2 g/kg. It is administered together with lemon or any other juice. It can be administered through a gastric tube to unconscious patients. Glycerol has a good hypotensive effect and can be given repeatedly. The substance is non-toxic and gives no side-effects. The metabolic activity of glycerol is high, it is a component of neutral fats and some phosphatides. Its anti-oedema efficacy does not depend on diuresis.

(e) In order to increase osmotic pressure in the plasma, albumin and hypertonic plasma solutions are administered. Their effect is rapid but not lasting.

2 It is very important to correct thoroughly the vital functions. Disorders in the cardiovascular and respiratory functions should be treated by symptomatic therapy. Prolonged intubation or tracheostoma for controlled lung ventilation are indicated in the presence of the smallest danger of asphyxia. The ventilation rate depends on the clinical picture and acid-base balance (pH,  $PCO_2$ ,  $PO_2$ , BE). The water-electrolyte metabolism should be corrected by administering various salt solutions, amino acids, protein, and other preparations.

3 Complex therapy of brain oedema includes hypothermia, during which the oxygen demand of cells decreases. Secondary oedema of the brain is absent in hypothermia, since it protects the brain in injury, vascular failure and surgical operations. The simplest way to attain hypothermia is applying cold (ice) to the head. Best results are attained with temperatures of 29-30°C. Hypothermia should be combined with neuroplegia. To that end, a 2.5 per cent aminazine solution, droperidol and other preparations should be used.

4 Complex therapy of brain oedema includes obligatory corticosteroids, which prevent development of oedema by normalizing permeability of the cerebral vessels. Maximum doses should be used to treat severe brain oedema: 5 mg/kg hydrocortisone and 2 mg/kg prednisolone.

5 The quantity of administered hypotonic solutions in brain oedema should be minimized. They should be administered with strict control of diuresis.

Signs of improvement do not indicate suspension of the therapy, because a relapse can occur at any time. Rational and timely therapy of brain oedema in children can ensure complete recovery without any sequelae (spastic and flaccid paralysis, disorders in the central nervous system). This is explained by the tremendous plastic properties of the cerebral cortex during its growth.

## Chapter 23

### Intensive Therapy of Hyperpyrexia

Hyperpyrexia is the elevation of the body temperature above  $39^{\circ}\text{C}$  causing severe disorders in the blood circulation and the central nervous system. These are manifested by deranged consciousness and symptoms of brain oedema. Hyperpyrexia occurs more frequently in infants and children than in adults, and it is more dangerous for children.

*Aetiology and pathogenesis* There are many causes by which temperature may rise, e.g. decomposition of red blood cells and labile thrombocytes, x-ray exposure, etc. Hyperpyrexia may occur after severe injuries with crushing of muscles and other tissues. Hyperpyrexia in such cases is due to the formation of auto-antibodies (decomposition of muscle proteins), which act by an allergic mechanism. Temperature can rise in processes causing pronounced immunohaematological shifts in the patient.

Upset balance of potassium and sodium ions also causes a rise in the body temperature. Hence, possible pyrexia following transfusion of salt solutions. Mechanical irritation of thermoregulating centres (fracture of the skull base, tumours in the hypothalamic region, etc.) often causes considerable hyperpyrexia. Temperature rises in neonates with intracranial haemorrhages, in post-encephalitic brain affections. The central disorder of thermoregulation is involved in these cases. Hyperpyrexia is most frequent in infectious diseases (acute respiratory diseases, pneumonia, measles, scarlet fever, influenza, dysentery, epidemic parotitis, etc.), in acute surgical diseases (appendicitis, peritonitis, osteomyelitis), it is connected with infection of the child with microbes and poisoning with their toxins. The temperature may rise significantly during post-operative periods in response of the child's body to the operative trauma. Pathogenesis of hyperpyrexia in children may be based on immaturity of the thermoregulating mechanism, insufficiency of the endocrine glands, and toxic affection of the central nervous system.

As the body temperature increases, the oxygen demands grow and the carbohydrate metabolism is upset. The glycogen level in the liver decreases due to intensification of glycogenolysis with rising temperature. Total cholesterol content of blood decreases (to 50 per cent of normal).

Infants, especially nurslings, often suffer from dehydration. The alkaline reserve in them decreases, while the amount of chlorine in the blood increases. The diuresis is low. The main bulk of water is removed from the body through respiration and perspiration. A single intake of water does not increase diuresis, since the liquid loss through the skin and respiration increases.

A rise of temperature by one degree causes an acceleration of the heart rate by 10 per minute. This is probably connected with intensifying metabolism and decreasing tone of the nervus vagus. Moreover, the blood flow-rate and pressure are changed. Arterial pressure falls, although in some cases it increases at elevated temperatures. Arterial pressure may increase due to disturbances in the central regulation, but hypertension is then followed by vascular hypotonia. Temperatures above  $41^{\circ}\text{C}$  are especially dangerous.

*Clinical picture* A sudden rise in body temperature is attended with flaccidity, adynamia and chill. The child refuses food and is thirsty. Sweating increases. Unless adequate therapy is administered in due time, symptoms indicating disturbances in the central nervous system develop. Motor and speech excitation develops along with hallucinations (mostly visual), clonicotonic convulsions occur. The child loses his consciousness, his eyes are fixed at a distance. Respiration is fast, shallow and irregular. Asphyxia (with a lethal outcome) may occur during convulsions. Children with the hyperpyrexia often develop circulatory disorders: the arterial pressure falls, the heart rate accelerates, and microcirculation is upset.

*Diagnosis* The diagnosis is not difficult. The vivid clinical picture supplemented with temperature measurements is quite conspicuous to exclude diagnostic error.

*Intensive therapy* The therapy is directed at correction of the vital functions and control of hyperpyrexia (Fig 51).

1 Temperature is lowered by physical and medicamentous means. Physical methods include cooling of the body by (a) removing clothes from the child, (b) applying ice packs to the head and groin where the femoral vessels closely underlie the skin, (c) treating the skin with alcohol, (d) irrigating the stomach (through a gastric tube) with water at a temperature of  $4-5^{\circ}\text{C}$  (this cools effectively the organs adjacent to the stomach, e.g. the liver, spleen and the abdominal aorta, through which to 50 per cent of the circulating blood volume is passed), (e) irrigation of the large intestine with ice-cold water, (f) blowing of air (from a fan), this decreases the body temperature by  $1-2^{\circ}\text{C}$  during 15-20 minutes.

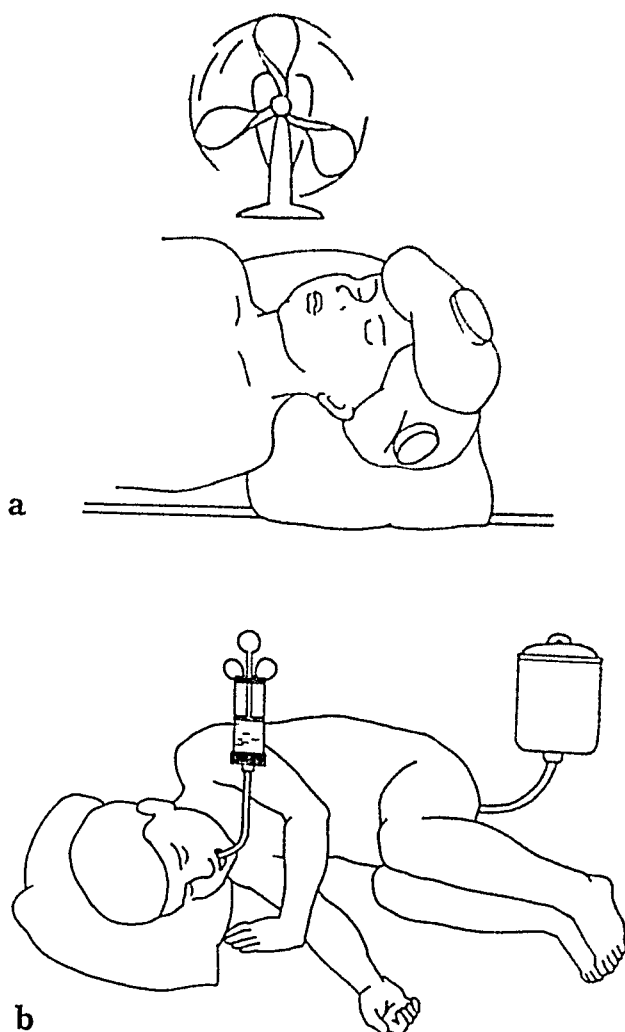
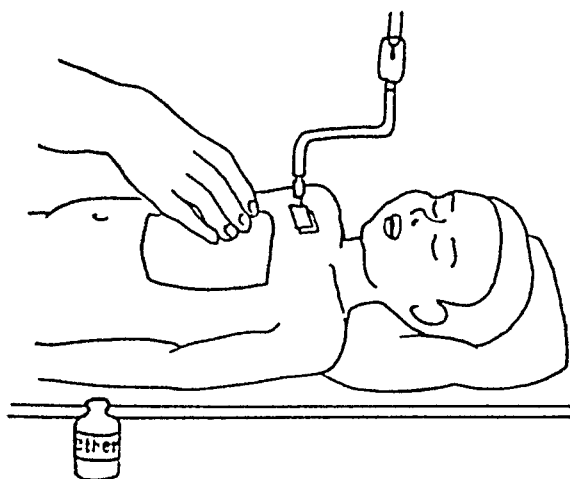


Fig 51 Intensive therapy of hyperpyrexia

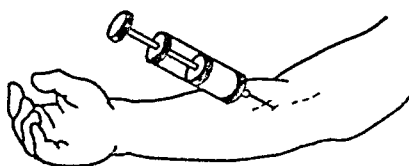
*a*—application of ice to the head, air blowing, *b*—administration of cold solutions into the stomach and rectum, *c*—sponging with alcohol or ether, *d*—administration of antipyretics

Some authors recommend cold baths and packs as physical methods of cooling. Of course, these methods can be used to treat hyperpyrexia, unless no emergency measures are necessary.

Analgin, amidopyrine, acetylsalicylic acid, aminazine, and some other antipyretics are used. Analgin should be administered in a dose of 0.1 ml of a 50 per cent solution per year of age. Amidopyrine is used in a dose of 1 ml of a 1 per cent solution per kg body weight. Acetylsalicylic acid is given in a dose of 0.05-0.1 g per year of age. Moreover, all solutions for infusion therapy should be cooled to 4°C. In our opinion, hyperpyrexia should be treated by complex therapy,



c



d

including both physical and chemical means. The temperature should not however be lowered below  $37.5^{\circ}\text{C}$  because after suspension of antipyretics therapy, the temperature continues decreasing by itself.

2. Correction of vital functions includes the following: (a) first of all it is necessary to quiet the child and to create a kind of neuroplegia. A 2.5 per cent aminazine solution (0.1 ml per year of age) is used for the purpose. In addition to anticonvulsive properties, aminazine has also a good antipyretic effect. Effective also are lytic mixtures which, in addition to aminazine, also contain pipolphen, which is given in the same doses as aminazine, (b) brain oedema should be eliminated by conducting dehydration therapy (see Ch 22), (c) in the presence of respiratory and cardiac dysfunction, intensive therapy should be directed at rapid elimination of these syndromes, (d) corticosteroids (hydrocortisone and prednisolone in doses of 3-5 and 1-2 mg/kg, respectively) should be administered to maintain adrenal function in the presence of hypotension, (e) hyperpyrexia is in most cases attended with metabolic acidosis, which can be corrected by administering a 4 per cent sodium hydrocarbonate solution. The necessary dose is calculated from the Smith formula, where base defi-



cit is multiplied by  $\frac{1}{2}$  body weight. If it is impossible to determine the acid-base balance, a 4 per cent sodium hydrogencarbonate solution can be administered in a dose of 0.1-0.2 g of dry substance per 1 g body weight.

## Chapter 24

### Intensive Therapy of Convulsions

The convulsive syndrome is a common one in children in critical conditions. This is associated with the period, anatomical and physiological characters of the child. Convulsions are involuntary muscular contractions, which occur suddenly, recurrently, lasting for various periods of time and indicating affection of the central nervous system.

*Aetiology and pathogenesis.* Convulsions may be caused by various harmful factors acting on the nervous system. Some of these factors can be the direct cause of convulsions, while others only act as provoking stimuli to indicate latent pathology of the central nervous system. The morphological and functional immaturity of brain in infants accounts for the very low threshold of excitation of the central nervous system and the tendency to diffuse reactions. Undetermined hydrophilic properties of the child's brain and increased vascular permeability are also predisposing factors. Various toxic and infectious factors account for the child's susceptibility to rapid development of brain oedema, which is manifested by convulsions. Various endogenic and exogenic factors arising in infections, intoxications, injuries, diseases of the central nervous system, and also psychogenic factors can precipitate convulsions in children. Convulsions occur in epilepsy, spasmodophilia, toxoplasmosis, encephalitis, meningitis, tumours of the brain, and in some other diseases. Convulsions sometimes occur in children with neurosis attended with increased excitability of the neuromuscular system. Metabolic disorders (hypoglycaemia, hypocalcaemia, acidosis) and endocrine dysfunction (adrenal insufficiency, pituitary dysfunction, and the like) can also be the cause of convulsions. Convulsions in neonates can be caused by asphyxia, haemolytic disease, and congenital defects of the central nervous system. Exposure to heat, recurrent vomiting and diarrhoea can sometimes cause convulsions as well. Convulsions associated with diarrhoea and vomiting are due to dehydration of the body and upset water-salt metabolism.

The triggering factor in asphyxia of neonates is oxygen deficit in the blood and tissues with accumulation of carbon dioxide and respiratory and metabolic acidosis. These factors interfere with normal circulation of blood, increase vascular permeability and produce brain oedema.

Convulsions in neonates with intracranial birth trauma are caused by intracranial haemorrhage, gliosis of the brain at sites of ischaemia, and subsequent atrophy of the brain tissues. Convulsions that develop after various injuries are also explained by haemorrhage and subsequent cicatrization and adhesion of tissues after its resolution.

Convulsions in infectious diseases are connected with infectious-toxic effect on the brain with subsequent development of intracranial hypertension and brain oedema. Children with bad allergic anamnesis have an increased susceptibility to convulsions. Convulsions developing along with skin eruption in children's infections indicate the phase of toxic encephalopathy.

Convulsions arising in prophylactic vaccinations develop by the mechanism similar to that observed in the antigen-antibody reactions, allergic predisposition of the body is often the provoking factor.

In acute neuroinfections convulsions are manifestations of general disorders in the brain, intracranial hypertension and brain oedema. Hypocalcaemia accounts for convulsions in spasmophilia.

*Clinical picture* Clinical manifestations of the convulsive syndrome are quite varied and depend on many factors. Convulsions differ in the time of their development, their duration, dependence on pain, degree of affection of the nervous system, the state of consciousness at the moment of convulsion onset, frequency of incidence, the amount of muscles involved, and the form of manifestation. Clonic convulsions are rapid muscular contractions which follow one another at short irregular intervals. Convulsions can be both rhythmic and arrhythmic and depend on the excitation of the cerebral cortex. Tonic convulsions are prolonged muscular contractions. They develop slowly and continue for long periods of time. Tonic convulsions can be primary, but can also be secondary to clonic (e.g. tonic convulsions develop immediately after clonic convulsions in epilepsy). Convulsions can be local and generalized. Tonic convulsions indicate excitation of subcortical brain nodes.

The clinical picture of the convulsive syndrome is quite specific. The child is suddenly abstracted from the surroundings, his eyeballs first move involuntarily and then are fixed in the upper or lateral position. The head is thrown back, the arms are bent in the elbows and wrists, the legs are stretched, and the jaws are closed. The tongue can be bit. The pulse and respiration are slow and apnoea is possible. This tonic phase of clonicotonic convulsions lasts not longer than a minute, and the child then takes a deep breath.

Clonic convulsions begin with muscular twitching of the face, the muscles of the limbs soon become involved and convulsions become generalized. Respiration becomes noisy, hoarse, and foam appears at the mouth. The skin is pallid. The heart rate is fast. Clonic convulsions vary in their length and can end lethally.

Various convulsions occur in children with various diseases. For example, tonic or clonicotonic convulsions occur in neonates with asphyxia, they disappear as soon as asphyxia is eliminated and brain oedema corrected.

Neonates with intracranial birth trauma usually develop generalized convulsions, tonic or clonicotonic contractions of the muscles attended by cyanosis and respiratory distress. The child can develop considerable hyperpyrexia. The anterior fontanelle is tense, the child suffers from vomiting and regurgitation. Convulsions occurring 2-3 months after birth can be epileptiform.

Convulsions occurring due to injuries are clonicotonic. The patient can be unconscious, vomiting, hemiplegia and affections of the cranial nerves can develop. Nystagmus, anisocoria, respiratory distress are possible too. They indicate compression of the cerebral stem.

Haemolytic disease of newborns is characterized by tonic convulsions which convert into opisthotonos. The child's face is distorted (Graefe's sign) and he shrieks.

The picture of an apoplectic stroke develops in acute disorders of cerebral circulation in children with septic processes. The patient is unconscious and clonic or clonicotonic convulsions are observed. Hemiplegia can be seen on the non-involved side.

In acute infectious diseases with affection of the central nervous system, convulsions develop at the height of the disease, they are tonic or clonicotonic. These convulsions are due to general cerebral disorders and indicate the encephalic reaction to microbial invasion. As a rule, these convulsions disappear with the fall of temperature.

Purulent meningitis is characterized by tonic convulsions in the extremities and clonic twitching of muscles of the face and trunk. Encephalitis is characterized by tremor, trismus and clonic convulsions during the initial stage of the disease.

As a rule, children with epilepsy are treated by neuropathologists and psychoneurologists. If an epileptic child with convulsions is admitted to the intensive therapy unit, he is treated as other patients with convulsions. It is necessary to take into consideration the previous treatment given to the patient and its efficacy.

*Diagnosis* Diagnosis is not difficult. But it is not sometimes easy to establish the cause of convulsions. A correct and thorough anamnesis helps in this situation. The health of the parents and information on the course of pregnancy (infections, intoxications, abuse of alcohol and medicines, exposure to occupational hazards, and threatening abortion) are especially important for correct assessment of the clinical picture of convulsions. It is necessary to find out the outcomes of the previous pregnancies and if there were pathological deviations during labour (intracranial injury, asphyxia, protracted labour). It is necessary to know the condition of the child before the onset of convulsions and the details of the first attack of convulsions.

In addition to anamnesis, it is also important to carry out additional examinations, such as electroencephalography. Encephalograms are characterized by reduced basic rhythm, hypersynchronous potential activity and peak-like oscillations. These paroxysmal signs on EEG, especially sharp waves and peak complexes with slow waves, indicate epileptiform pathology.

Skull x-ray is also important for diagnosis. The presence of changes in the bone structure (with characteristic clinical picture) can serve as organic cause of convulsive attacks.

Rheoencephalography can be used to assess filling of the blood vessels and the condition of the vascular wall, and to reveal asymmetry in blood filling of various portions of the brain. These changes in infants can be due to congenital growth defects or the results of perinatal pathology, they can be the cause of epileptiform attacks.

Cerebrospinal puncture is diagnostically important. Increased pressure in the cerebrospinal fluid (above 130 mm H<sub>2</sub>O) indicates liquor hypertension. The pressure can remain normal in the presence of hypertension only in cases where the liquor routes are blocked. If the liquor ducts are blocked above the point of puncture, the liquor pressure does not rise in response to compression of the jugular veins. Blockage of the subarachnoid space in the lower thoracic and lumbar regions does not increase the liquor pressure when pressure is applied to the abdominal vein region for a few seconds. The presence of blood, fresh or leached erythrocytes in the liquor indicates subarachnoid haemorrhage. Normally, 1 cu mm of liquor of infants contains from 5 to 20 cells, 0.2-0.3 per cent of albumin and 50-60 mg% of sugar. Increasing number of cells in the cerebrospinal fluid and lymphocytic pleocytosis indicate serous meningitis. Cloudy fluid, neutrophilic or mixed neutrophilolymphocytic pleocytosis, and also increasing amount of albumin suggest purulent meningitis. Increasing concentration of albumin in the liquor (in the presence of altered but relatively stable pleocytosis) indicates blockage of the liquor ducts. The presence of protein-cellular dissociation, i.e. increasing concentration of protein in normal cytosol, can suggest the presence of new growth.

An important additional method is examination of the fundus oculi. Asymmetric haemorrhages in the retina indicate intracranial haemorrhage. Congestive papillae of the optic nerve indicate increased intracranial pressure. Inflammatory diseases in the cranium are manifested by neuritis of the optic nerve.

It is always necessary to suspect connection between convulsions and infectious-inflammatory diseases, changes provoked by disordered water-electrolyte and mineral metabolism, intoxication caused by disturbances in the detoxicating function of the kidneys and liver. This will ensure a correct pathogenetic approach to treatment of such patients, in addition to symptomatic therapy.

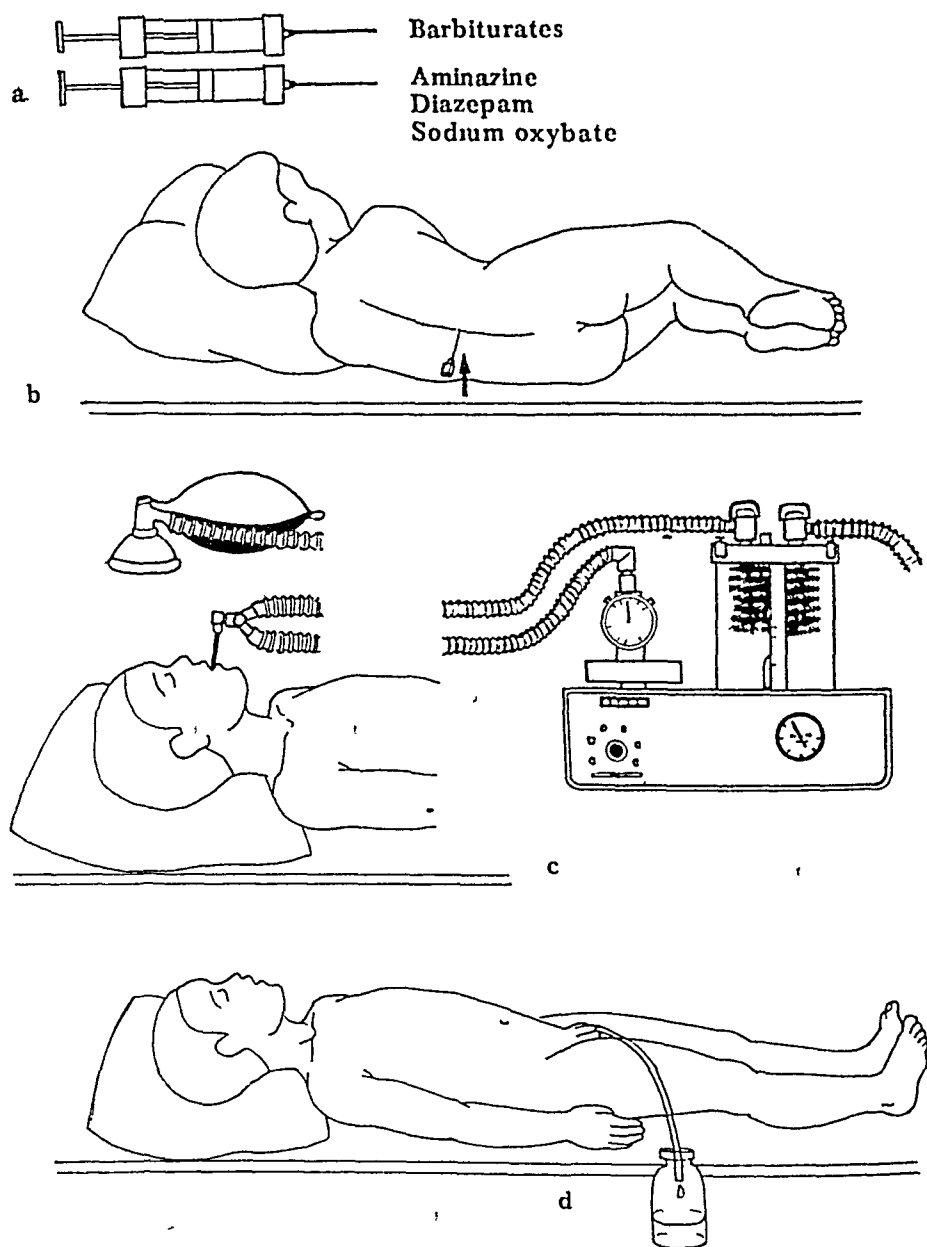


Fig. 52 Intensive therapy of convulsions

a—administration of anticonvulsants, b—cerebrospinal puncture, c—anaesthesia and artificial lung ventilation, d—dehydration

*Intensive therapy* The intensive therapy of convulsions includes the following correction and maintenance of vital body functions, anticonvulsive and dehydration therapy (Fig 52) The sequence of procedures depends on the child's condition It is quite evident that

if convulsions occur in the presence of marked disorders in respiration, blood circulation and water-electrolyte metabolism, which are a direct danger to the child's life, intensive therapy should be begun with correction of these functions. The anticonvulsive therapy is often combined with correction of the vital functions.

1 Maintenance of the vital body functions includes the following (a) removal of any hindrances in the airways by aspiration, (b) oxygen therapy, (c) artificial ventilation of the lungs (whenever necessary), (d) maintaining normal circulation of blood, (e) control of the water-electrolyte metabolism, acid-base balance, and other biochemical indices of homeostasis (any shifts should immediately be corrected), (f) administration of hormone preparations.

2 Anticonvulsive therapy is carried out by various methods and using various medicines. The methods are described in the order of their increasing efficacy. It should be understood that it is unnecessary to use all means available. The therapy given in each particular case depends on the skill of the resuscitator and the child's condition. (a) Rectal administration of 1-3 per cent chloral hydrate solution (0.2 g of dry substance per year of age). Chloral hydrate has an adverse effect on the respiratory centre and it should not therefore be used in the presence of respiratory disorders, (b) intramuscular or intravenous administration of aminazine in a dose of 1-2 mg/kg. Aminazine should preferably be administered together with pipolphen, (c) short-acting barbiturates should be infused slowly until convulsions are eliminated. Barbiturates include a 2 per cent hexenal or a 1 per cent thiopental sodium. As a rule, 3-5 ml of the solution will be sufficient, the preparations can be administered again in recurrent attacks, (d) sodium oxybate (20 per cent solution) is a very effective anticonvulsive and hypnotic preparation. It should be infused rapidly (1 ml per year of age). Sodium oxybate can be administered by drip injections in a 5 per cent glucose or isotonic sodium chloride solution (to prevent repeated convulsions). If convulsions should be removed immediately, nitrous oxide with oxygen (3:1) should be given for inhalation anaesthesia in combination with halothane. This method should however be used only if other methods fail. If anaesthesia does not remove convulsions, which cause respiratory distress, muscle relaxants should also be administered and the lungs ventilated artificially.

3 Dehydration therapy is obligatory, it includes the following (a) magnesium sulphate, a 25 per cent solution (1 ml per year of age). The injection can be intravenous in severe cases. A 10 per cent calcium chloride solution should be infused in a dose of 1 ml per year of age, 10-20 ml of a 40 per cent glucose solution with vitamins B and C should also be infused, (b) lasix should be injected intravenously or intramuscularly in a dose of 3-5 mg/kg a day, (c) mercury diuretics (novurit, fonurit) should be administered in a dose of 0.1 ml per year.

of age, (d) glycerol, which is an effective dehydrating agent, should be administered into the stomach through a tube in a dose of 5-15 ml, (e) if convulsions persist, the use of osmotic diuretics should be considered a 15-30 per cent solution of mannitol (5-10 ml/kg) or urea (1-1.5 g/kg) should be used

4 If these measures fail, lumbar puncture should be done to withdraw 5-10 ml of liquor

## Chapter 25

### Infusion Therapy of Water-electrolyte Disbalance

Infusion (infusion-transfusion) therapy is the method of parenteral administration of various substances distributed in an aqueous phase. Infusion therapy is directed at maintaining the main body functions and biochemical processes. Infusion therapy is used to correct or maintain (within the required range) the volaemic status (volumes of body fluids), hydrogen ion concentration, and acid-base balance, it improves the properties of blood, ensures detoxication of the body, passive immunization, supply of the body with plastic and energy substrates, and also parenteral administration of medicines at a strictly defined rate. These problems can be solved either separately, in sequence, or simultaneously.

**Methods of infusion therapy** At the present time the only effective method of infusion therapy is intravascular administration of solutions. In most cases the solutions are administered intravenously, although intra-arterial infusions are also used. In exceptional cases (when other methods fail) the solutions can be administered intraosseously, but only a limited number of solutions can be administered by this method. Any suitable vein can be used for infusion of solutions. Conditions permitting, the site and approach to the vein should be selected depending on the volume of infused solution, intensity and duration of infusion therapy (Figs 53-56, Plate 9).

**Solutions for infusion therapy** Modern classifications of solutions for infusion therapy lack terminological unanimity and have some other disadvantages. We shall therefore only name these solutions and specify indications for them.

**Blood preparations** These include whole (canned) blood, plasma, erythrocytes, leucocytes, thrombocytes, etc. They are indicated for hypovolaemia of any aetiology, anaemia, hypoalbuminaemia, leucopenia, thrombocytopenia (not associated with disseminated intravascular coagulation), decreased coagulability of blood, and some other conditions.

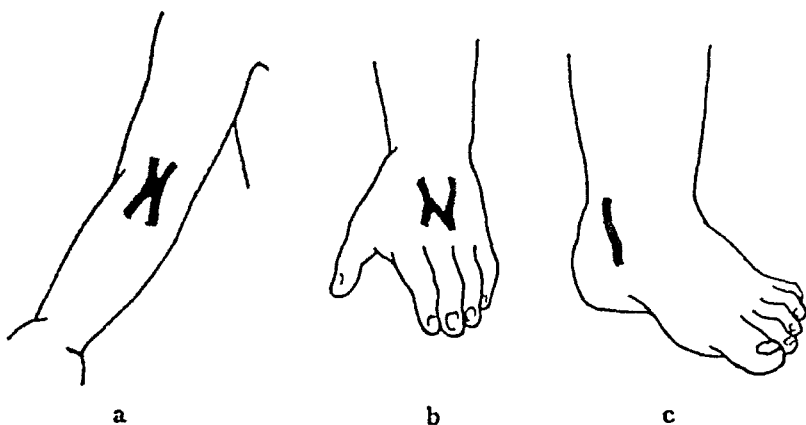


Fig 53 Peripheral veins suitable for puncturing  
*a*—cubital vein, *b*—veins of the hand, *c*—anterior vein

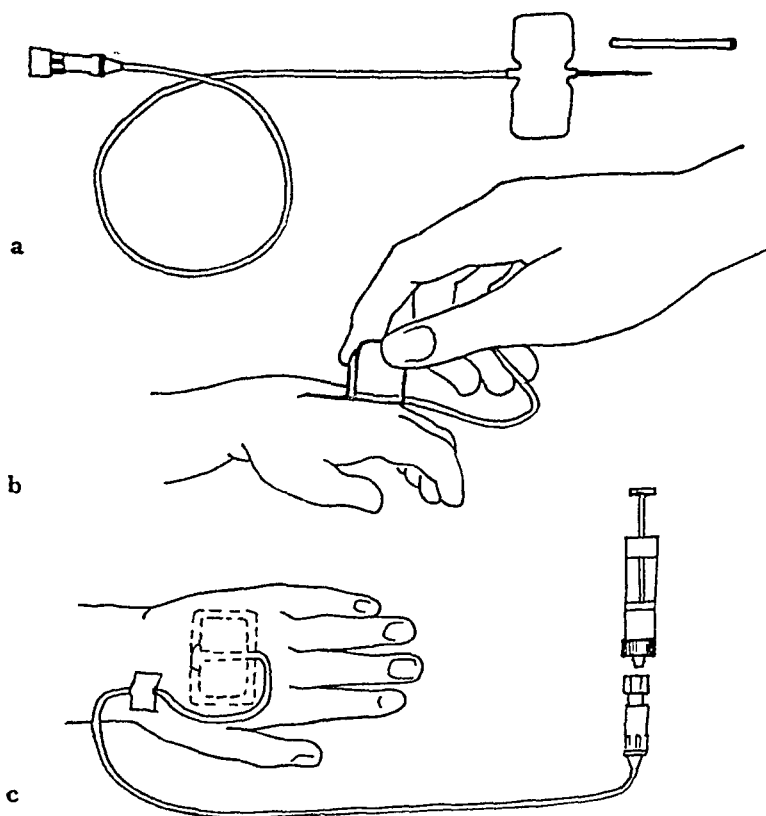


Fig 54 Vein puncturing and fixation  
*a*—winged needle device, *b*—puncturing the vein, *c*—vein fixation



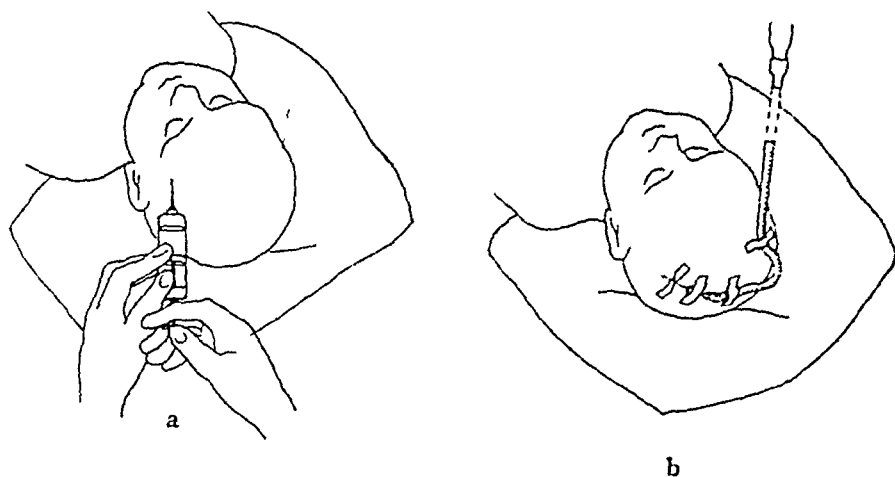


Fig 55 Puncturing (a) and fixing (b) head veins

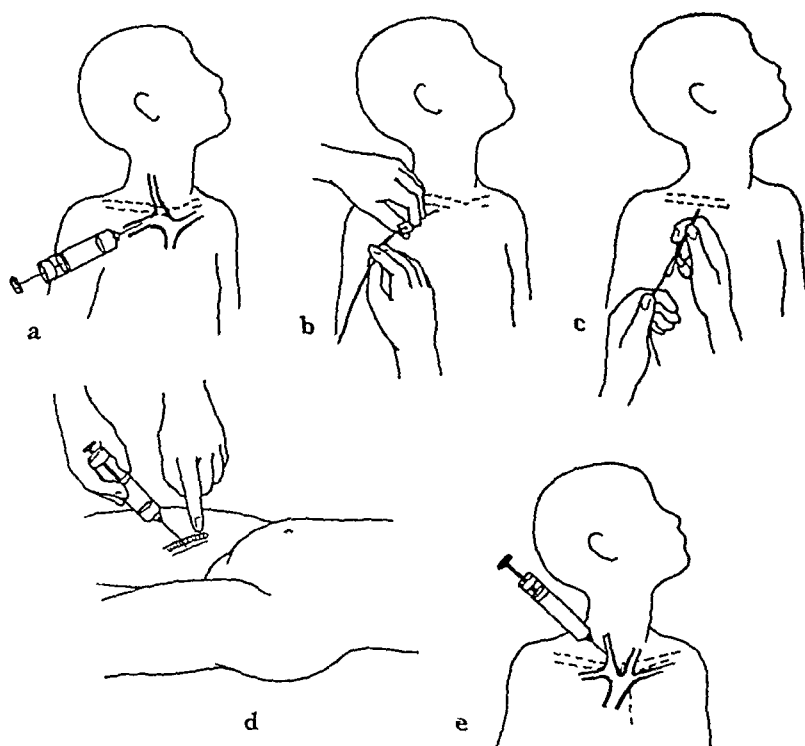


Fig 56 Cannulation of the main veins according to Seldinger

*a*—puncturing subclavian vein, *b*—passing a guide wire through the needle, *c*—passing a catheter over the guide wire into the vein, *d*—puncturing a femoral vein, *e*—puncturing internal jugular vein

*Preparations for replenishment of the circulating blood volume.* These preparations are often used in anaesthesiology, intensive therapy and resuscitation because hypovolaemia, associated with the loss of plasma, frequently occurs in various pathological conditions in children. Preparations of this group include natural and synthetic solutions, such as plasma, albumin solutions, protein, polyglucin, rheopolyglucin, polyvinol, and others. Ringer's and Hartmann's solutions have been recently proved to be quite effective in correcting hypovolaemia. Water deficit can be corrected using 5 and 10 per cent glucose solutions.

Isotonic sodium chloride solution and polyionic solutions of various composition are widely used in vomiting, diarrhoea, gastrointestinal draining, and in other conditions to replenish pathological loss of fluids containing ions.

Concentrated solutions of sodium chloride (10 and 5 per cent solutions), potassium (7.5 per cent solution), calcium (10 per cent solution), magnesium sulphate (25 per cent solution) are used to meet normal demands for the main cations or to eliminate their deficit. As a rule, the solutions are administered uniformly and as slowly as possible. But in some cases it is necessary to infuse solutions at a fast rate. For example, in order to eliminate severe hyponatraemia in the presence of dehydration, a 10 per cent sodium chloride solution is infused at a fast rate, magnesium and calcium solutions can also be infused at a fast rate.

Potassium chloride may be infused only in concentrated (10-15 per cent and more) glucose solutions with insulin, while the concentration of potassium chloride proper should be not higher than 1 per cent. The daily dose (in the absence of potassium deficit) should not exceed 3 mmole/kg for infants to 6 months of age and 2 mmole/kg for older infants. The daily potassium dose should be administered in 3-5 portions during the course of 16 to 24 hours. The safe potassium concentration in the solution is 40 mmole/l (4 ml of a 7.5 per cent solution of potassium chloride in 96 ml of glucose solution). In the presence of potassium deficit, the concentration can be increased to 80-150 mmole/l with strict control of potassium level in the plasma and erythrocytes. Administration of potassium in oliguria is contraindicated.

In order to remove hyperhydration, osmotically active substances, such as sorbitol or mannitol, should be used in solutions of considerable concentration. The solutions should be infused at a considerably high rate in order to provide a considerable increase in osmotic concentration of a given substance in the serum, which in turn induces osmotic diuresis.

Acid-base disbalance in metabolic acidosis is corrected with 4 and 8 per cent sodium hydrocarbonate solutions, less frequently with tris-buffer and trimethyl aminomethane solutions. Large doses

of potassium chloride are most effective in metabolic alkalosis, especially in post-aggressive conditions

Detoxicating preparations are often used for intensive therapy of children with various infectious processes. These preparations include substances capable of adsorb toxic products of decomposition of bacteria and of own cells of the body. Haemodes, albumin, plasma, protein, rheopolyglucin and the like preparations are believed to be most effective.

Preparations and solutions for parenteral nutrition form a special group, which can be divided into the following three subgroups: preparations for plastic, energoplastic, and energy supply. The preparations of the first type include protein hydrolysates and solutions of crystalline amino acids, free of alcohol or carbohydrate. Preparations of the second type contain solutions of crystalline amino acids containing energy substrates, such as sorbitol, xylitol, fructose, and less frequently glucose and ethanol. Glucose solutions (concentration from 10 to 70 per cent) and fat emulsions are energy substrates. It should be noted that part of carbohydrates and fats is involved in the building up of structural elements of cells and tissues of the body. Preparations for parenteral nutrition include solutions of vitamins and microelements.

**Infusion therapy.** Infusion therapy during surgical operations, intensive therapy and resuscitation of infants and children includes a series of consecutive operations and appropriate calculations.

The principles of infusion therapy are very simple. They are (a) meeting normal physiological demands of children for water and ions, (b) correction of water or ion deficit, (c) replenishment of current pathological loss of water and ions.

The first principle is realized by administering water and ions to meet the normal physiological demands. These quantities can be calculated by several techniques: (1) gravimetrically— $V_{ml/s} = \text{dose in ml/kg} \times s$ , (2) calorimetrically— $V_{ml/s} = 100 \text{ ml/100 kcal} \times \text{energy demand (kcal)}$ , (3) by the surface area of the body— $V_{ml/s} = 2000 \text{ ml/m}^2 \times S \text{ m}^2$ .

Correction of water and ion deficit is a difficult problem because it is difficult to determine accurately the lacking amounts of water and ions, especially in older children. The simplest method to determine the deficit of water is by calculating the difference of the child's body weight before the onset of the disease and at the moment of examination. The difference is the liquid deficit. But this method is only effective in acute disorders of water-salt metabolism or if the weight of the child before the onset of the disease is known. This method is inapplicable to older children, especially to children whose anthropometry differs significantly from average indices (normal for the given age) and to children whose weight is unknown. The liquid deficit can in such cases be determined by the change of the

haematocrit using the following formula

$$\frac{Ht_p - Ht_n}{100 - Ht_n} \times b \text{ wt} \times K$$

where  $Ht_p$  is the haematocrit of the patient,  $Ht_n$  normal haematocrit for the child of a given age,  $b \text{ wt}$  is the body weight in kg, and  $K$  is the coefficient, which is 0.3 for infants under 1 year and 0.2 for older children. It should be noted that the error of determination of liquid deficit by this formula can be great, and this formula is therefore rarely used in practice.

The deficit of ions can be determined also using special formulas with due consideration of the error. The deficit of the extracellular sodium can be found using this formula

$$(140 - C_p) \times b \text{ wt} \times K = \text{deficit, in mmoles}$$

where  $C_p$  is the concentration of sodium in the serum of the child and  $K$  is the same coefficient as in the previous formula. The deficit of the intracellular potassium can be determined from the formula

$$(80 - C_p) \times b.\text{wt} \times 0.4 = \text{deficit, in mmoles}$$

where 80 is the lower limit of normal concentration of potassium in erythrocytes,  $C_p$  potassium concentration in the child's erythrocytes, and  $b \text{ wt} \times 0.4$  is the volume of liquid in the intracellular space.

The accuracy of the formulas can be slightly improved if the coefficients are replaced by the relative volumes of liquids in the intra- and extracellular space as specified for various ages in Ch. 3. It should be noted that the ion deficit rarely occurs without changes in the water content of the body, and this deteriorates the accuracy of calculations.

The replenishment of current pathological loss is not difficult. The volume of pathologically lost liquid (except liquid loss with perspiration) can be measured. The concentration of the main ions in the lost liquid can also be determined, and this enables the realization of the third principle of infusion therapy to a sufficiently high degree of accuracy.

The pathological loss of water with perspiration can be replenished as follows: when the body temperature increases in the absence of marked dyspnoea, 10 ml/(kg  $\times$  day) of water should be added to the total daily given volume of water per degree centigrade above 37°C, on condition that the elevated temperature persists for 24 hours. If the fever is shorter, the additive should be decreased correspondingly. In the presence of dyspnoea, another 10 ml/(kg  $\times$  day) should be added per each ten respiratory cycles.

Pathological loss of liquid from the gastrointestinal tract should be replenished by two methods. One of them is very tentative. It

consists in administration of the ion solutions (usually Ringer's solution) whose volume is equal to the liquid volume lost. A more accurate method is administration of a 5 per cent glucose solution containing concentrated solutions of ions. The quantity of ions should be equal to the quantity of lost ions, which should be determined by analysing the lost liquid and calculating the total quantity of the ions lost. It is reasonable to replenish pathological losses during infusion therapy, by adding the appropriate quantity of water and ion solutions to the daily infused volumes (at 6-8-hour intervals).

The total quantity of water and ions, which the child should obtain with infusion therapy, should be equal to the sum of physiological demands for water and ions, the deficit (if any), and pathological losses. If the child is able to eat and drink, possible or factual amounts of liquid taken as food should be taken into consideration during calculation of infusion therapy volumes. The quantity of liquid that should be infused intravenously should be decreased correspondingly (by the volumes that are taken with food). The quantity of infused ions can be left unchanged.

An important factor of infusion therapy is the rate of infusion. It can be rather high when liquids are infused for hypovolaemia. The dose should be infused within 90 minutes maximum. In the presence of haemorrhage, plasmorrhoea (from injured skin), haemorrhagic or hypovolaemic shock, the rate of infusion should be very high. When liquid deficit is being eliminated, it is not recommended to replenish the entire liquid loss within 24 hours, especially if dehydration is severe (exceeding 6-7 per cent). The deficit should be better replenished during two days,  $\frac{2}{3}$  deficit being replenished during the first day and  $\frac{1}{3}$  during the second day.

A simple way to regulate the infusion rate is by the number of drops per minute. For most dropping systems and substances the size of a drop is practically 0.05 ml. It follows therefore that one millilitre contains 20 drops.

The formula for the determination of the hourly infusion rate is derived as follows. The minute infusion rate being 10 drops, the hourly infusion rate is  $10 \text{ drops} \times 60 \text{ min} \times \text{hr}/20 \text{ drop/ml} = 10 \times 3 = 30 \text{ ml/hr}$ . The factor of 3 is used to determine the hourly infusion rate in millilitres. These relationships are used to determine the volume of liquid infused during an hour in  $\text{ml} = \text{drop/min} \times 3$ , and the rate of infusion in  $\text{drop/min}$  during an hour

$$V_{\text{drop/min}} = \frac{\text{ml/hr}}{3}$$

$$V_{\text{drop/min}} = \frac{N \text{ ml}}{T_{\text{hr}} \times 3}$$

The time during which the known volume of liquid can be infused at a given rate in drop/min is

$$T_{\text{hr}} = \frac{1 \text{ in ml}}{\text{drop/min} \times 3}$$

The volume of one portion of solution for drip infusion to infants under 12 months should not exceed  $\frac{1}{4}$  of daily volume

**Correction of acid-base balance.** The correction of acid-base disbalance, in particular respiratory disorders, is not the prerogative of infusion therapy. Therefore, in this section we shall only consider disorders that cannot be eliminated by means other than infusion therapy, or at least by administration of some solutions. Metabolic acidosis and metabolic alkalosis are such disorders.

*Elimination of metabolic acidosis.* We have already named the causes of metabolic acidosis. This condition can to a certain degree aggravate the course of the pathology responsible for the onset of metabolic acidosis (thus to complete the vicious circle). In order to eliminate metabolic acidosis the following should be done: (1) hypovolaemia should be eliminated by any possible means, (2) perfusion of peripheral tissues should be improved, (if removal of hypovolaemia fails to give the desired effect, vasoplegics, or less frequently, vasoconstrictors should be used), (3) oxygen supply to the child should be ensured by all possible means of oxygen therapy (artificial ventilation included), (4) the contractile power of the myocardium should be strengthened by medicines, if necessary, (5) adequate diuresis should be ensured, (6) supply of energy substrates (carbohydrates, sometimes with insulin) should be ensured, (7) vitamins B should be administered in therapeutic doses.

This complex of measures is effective practically in all cases except where sufficient blood oxygenation in the lungs is unfeasible.

Sodium bicarbonate should be administered if the pH is as high as 7.1 or 7.0. This may be necessary during resuscitation or if the bicarbonate is lost with the material evacuated from the lower portions of the small intestine. It should be remembered that except in the last case, administration of sodium bicarbonate is a palliative (non-pathogenetic) measure, which does not eliminate the cause of acidosis.

*Elimination of metabolic alkalosis.* It has been said that metabolic alkalosis most often occurs after critical conditions due to potassium loss from the child's body. As 3 potassium ions leave the cell, they are replaced by 2 sodium ions and 1 hydrogen ion. This causes the hydrogen ion deficit in the extracellular fluid and alkalosis in the cell. Metabolic alkalosis is eliminated by administering big doses of potassium chloride simultaneously with energy substrate (usually, glucose added by insulin). In these cases the glucose concentration is not less than 10-15 per cent. The time during which meta-

bolism can be eliminated depends on the degree of potassium deficit. As a rule, it takes not less than 36 hours. The potassium concentration in the infusion solution can be 100 mmole/l (10 ml of a 7.5 per cent potassium chloride solution in 190 ml of solution). In some cases the potassium concentration can be even higher (150 mmole/l), but the ion concentration in the plasma and erythrocytes should in such cases be determined in the laboratory at short intervals, cardiomonitoring is also required. Elimination of potassium deficit and metabolic alkalosis should not be begun before the complete recovery of the renal function. Efficacy of treatment can be controlled by the potassium concentration in the intracellular fluid of erythrocytes and the pH of the urine. If the urine reacts alkaline (the pH is determined in fresh urine) it indicates efficacy of the therapy. Elimination of the deficit corresponds to normalization of the acid-base balance and potassium concentration in the intracellular fluid of erythrocytes.

*Haemorrhagic shock* According to modern views, canned blood, Hartmann's solution (Ringer lactate) and synthetic plasma expanders are the best preparations to treat haemorrhagic shock. The treatment usually begins with administration of plasma expanders or the Ringer lactate solution until the group of blood and the Rhesus factor of the patient are determined and the donor blood is tested for compatibility with the patient's blood. The rate of infusion is controlled by the arterial pressure, pulse rate and the appearance of the urine. If the patient is in a severe shock, all preparations can be administered simultaneously into different veins. It is recommended to administer a polarizing mixture (20 per cent glucose solution, potassium in the concentration of up to 40 mmole/l, insulin in a dose of 1 U per 2-3 g of dry glucose).

*Hypovolaemic shock* This usually occurs in dehydration or abrupt fall of the vascular tone. The therapy is actually the same as in the previous case, except that blood transfusion is unnecessary. The main preparations are plasma expanders of any type (synthetic, e.g. polyglucin or rheopolyglucin, homogenous or isogenous, e.g. albumin, plasma, Ringer lactate).

*Hypotonic dehydration* Severe disorders in the water-salt metabolism frequently combine with pronounced hyponatraemia. Treatment should begin with partial correction of the sodium deficit. This can be done by administering 0.5-1 ml/kg of a 10 per cent sodium chloride solution. Further treatment should be conducted in accordance with the general principles, paying special attention to the primary elimination of hypovolaemia, which practically always attends this type of disorder.

*Hypertonic dehydration* Solutions that do not contain sodium ions should not be administered even during the early stage of treatment. When solutions are infused at a fast rate, the ratio of solutions con-

taining glucose and sodium should be about 5:1. If infusion is slow, the ratio is 4:1.

*Renal failure* The volume of solution that should be infused for acute renal failure should be determined by the sum of fluid volumes lost with perspiration and excreted with urine. The volume of liquid to be infused in anuria should correspond to the liquid loss with perspiration, less water of oxidation.

## Chapter 26

### Parenteral Nutrition in Intensive Therapy

Parenteral nutrition is part of infusion therapy. It means the supply of all necessary nutrients that cannot be ingested per os. The main indication for parenteral nutrition is impossible feeding of a child by any other method. Sometimes nutrition is given parenterally in cases where normal feeding or administration of food through a gastric tube does not meet the body demands for nutrients.

*Classification* Parenteral nutrition can be total, partial, or additional.

*Total* parenteral nutrition (hyperalimentation) means intravenous administration of the total nutrient requirements.

*Partial* parenteral nutrition is intravenous administration of the lacking amounts of all required nutrients in addition to administration of nutrients by other routes (per os or through a gastric tube).

*Additional* parenteral nutrition is intravenous administration of some nutrients, for which the child's body has increased demands.

From the point of view of biochemistry, the main difference of parenteral nutrition from ordinary one is that parenteral nutrition does not require transformation of food polymers into monomers (amino acids, hexose) except hydrolysis of neutral fat, which is a part of fat emulsions. Fats are hydrolysed in the blood vessels by the action of lipoprotein lipase of plasma. The intracellular metabolism of nutrient monomers, administered either parenterally or per os, is otherwise the same.

*Parenteral nutrition systems* We shall consider two main systems of parenteral nutrition. One of them is used to administer all required nutrients, amino acids, carbohydrates (glucose), and fats. Fats are not administered parenterally with the other system and all energy requirements are met by carbohydrates alone. In the latter case, the glucose dose should exceed the normal demand two times.

During recent years fat emulsions are produced for parenteral nutrition. The system of balanced parenteral nutrition is therefore more frequently used now.



**Planning total parenteral nutrition** Calculating the daily programme for total parenteral nutrition is similar to planning the daily infusion therapy programme. First of all, the total volume of liquid that can be infused to a given child during 24 hours should be determined. This volume depends on the condition of the child, his cardiovascular and excretory functions, and some other factors. The greatest difficulties are met when making out the programme for parenteral nutrition of children in critical conditions, because in addition to parenteral nutrition, they should also be given infusion therapy.

The daily parenteral nutrition programme is calculated with reference to the daily demands of the child for nutrients and the volumes of solutions and preparations for parenteral nutrition. The daily demands for the main ingredients of parenteral nutrition for children of various age are given in Table 25.

Once the concentration of solutions and emulsions for parenteral nutrition, and the daily demands for fat, protein, and carbohydrates are known, the volumes of these preparations can be calculated. It should be remembered that 1 g of fat is equivalent to 9 kcal, 1 g of protein 4 kcal, and 1 g of carbohydrates 4 kcal. A well balanced parenteral nutrition satisfies the energy demands to 50 per cent by carbohydrates, to 40 per cent by fats, and to 10 per cent by proteins. In hyperalimentation 90 per cent of the energy demands are satisfied by carbohydrates and 10 per cent by proteins. The required ingredients are infused in the volume of liquid equal to the daily liquid demand. The nitrogen to energy ratio of the parenteral nutrition should be not less than 1 g 200 kcal. Otherwise the administered amino acids will oxidize with the release of the lacking amount of energy.

**General instructions.** Solutions and preparations for parenteral nutrition can be administered at any point of the blood circulating system. Hyperalimentation should preferably be conducted through catheters inserted into the central veins because solutions of high osmotic concentration, which are used in such cases, can damage the venous intima. Large vessels are less vulnerable to this effect. If peripheral veins are used for the purpose, a necessity arises to change the vein frequently. If total parenteral nutrition is given, any vein can be used, but practical experience shows that the central veins should be preferred.

All ingredients of parenteral nutrition should be administered simultaneously. The protein preparations (solutions of crystalline amino acids, protein hydrolysates) should be mixed with carbohydrate solutions in one vessel. Fat emulsions should be administered simultaneously with the other ingredients but from a separate dripping system. The systems are joined into one before the entrance into the blood vessel. Fat emulsion should not be mixed with any other solution or preparation!

Table 25 Normal Daily Nutrient Demands

| Ingredient                    | Age     |         |         |         |         |         |         |         |
|-------------------------------|---------|---------|---------|---------|---------|---------|---------|---------|
|                               | Neonate | 1-6 m   | 6-12 m  | 1-3 y   | 3-6 y   | 6-9 y   | 9-12 y  | 12-15 y |
| Water, ml/kg                  | 100-150 | 130-160 | 120-150 | 135-140 | 110-90  | 90-75   | 85-65   | 65-40   |
| Energy, kJ/kg $\times$ s      | 377-502 | 356-502 | 335-449 | 335-419 | 335-377 | 314-314 | 293-335 | 251-293 |
| Protein, g/kg                 | 2 4-4   | 1 85-4  | 1 2-4   | 1 2-4   | 1-3 5   | 0 88-3  | 0 8-2 5 | 0 7-2   |
| Glucose, g/kg                 |         |         |         |         |         |         |         |         |
| total parenteral nutrition    | 12-18   | 12-18   | 12-18   | 15-16   | 14-15   | 12-13   | 12-10   | 8 10    |
| hyperalimentation             | to 30   | to 30   | to 27   | to 27   | to 25   | to 25   | to 24   | to 20   |
| Fat (total parent nutr), g/kg | 4       | 4       | 4       | 3       | 3       | 2       | 2       | 1-1 5   |

*Note* Protein doses are given in the range between safe intake of high-quality proteins (eggs, milk) and total protein intake, which is assessed as 70 per cent of above standard (eggs and milk) preparations used for nutrition are prepared from high-quality proteins or their composition approaches the composition of standard protein (solutions of crystalline amino acids)

Parenteral nutrition should be conducted as long as possible, normally during 22-23 hours a day

During hyperalimentation it is necessary to increase gradually the glucose dose by increasing its concentration in solution (during 2-5 days) until a full dose is administered. Parenteral nutrition should not be interrupted for more than 30-60 minutes, especially with full carbohydrate dose. The infusion therapy should continue for not less than 2-4 days.

**Control of parenteral nutrition** The following should be controlled during parenteral nutrition: correct filling of the system, thorough connection of the catheters to the administering system, cleanliness of the dripping system, correct storage of solutions and preparations, the rate of their administration, and other parameters. Biochemical indices of the acid-base balance, haematocrit, haemoglobin concentration in the blood, concentration of plasma albumin, urea, glucose, the main ions of the serum, transaminase and bilirubin should also be controlled.

The chylous properties of serum and also the concentration of non-esterified fatty acids, triglycerides and cholesterol in the serum should also be determined. Moreover, in addition to these indices, it is also necessary to control some other factors that are important for common infusion therapy, such as the daily diuresis, concentration of the main ions in serum, etc.

The regularity of control depends on the value of the obtained information in each particular case and on the child's condition prior to the administration of parenteral nutrition. The determination of various indices should be more frequent during the initial period of parenteral nutrition, but later it may be carried out once or twice a week.

**Complications of parenteral nutrition** When administering parenteral nutrition, the physician may encounter with various complications. Most of them are due to incorrect assessment of the patient's condition, errors in calculation of the amounts of nutrient ingredients, incorrect technique of administration of nutrition, and inadequate control of the patient's condition. In other words, practically all complications are iatrogenic in their aetiology. All complications of parenteral nutrition can be classified as technical, metabolic and infectious.

*Technical* complications include those, which arise due to improper catheterization of the vessels, incorrect use of various means of infusion therapy (dripping systems, perfusion pumps, etc.)

*Metabolic* complications are manifested by marked changes in various types of metabolism. They can sometimes have quite specific clinical picture. Metabolic complications include hyper- and hypoglycaemia, osmotic diuresis, dehydration, hyper- and hypocalcaemia, hyper- and hypophosphataemia, deficit of essential fatty acids,

hyperammonemia, increased activity of seral transaminase and concentration of bilirubin, cholestatic jaundice, etc. Prevention of these complications depends on thorough daily observation of the child's condition and strict observation of all rules for correct total parenteral nutrition. The complications can be removed by analysing the prior changes in the child's condition, previous course of parenteral nutrition, the current daily nutrition programme, with subsequent alteration of the nutrition conditions within the current or next day.

*Infectious* complications include adverse effects of bacterial or fungal flora on the child, which are mainly due to administration of total parenteral nutrition. These complications can vary in form and intensity, from insignificant inflammatory reaction of the skin around the point of insertion of the venous catheter to sepsis. In most cases these complications are due to inadequate care of the parenteral nutrition system (infrequent change of the bandage fixing the catheter, disjoining of various connectors in the system, soiling the system with blood, solutions and preparations for parenteral nutrition, neglect of aseptic rules during mixing the solutions or preparations, etc.) Another cause of infectious complications is infection of the child prior to parenteral nutrition. It has been noted that the incidence of sepsis during parenteral nutrition is rather high, in children and adults, it increases with the duration of the course.

Prevention of infectious complications depends on thorough observation of all rules of prolonged and massive infusion therapy and parenteral nutrition.

**Efficacy of parenteral nutrition.** Efficacy of parenteral nutrition is not questioned in general, but it is difficult to assess the daily effect of this nutrition in a given child. Unfortunately, there are no accurate means of assessment except by the nitrogen balance. But the technique is very difficult and it is rarely used in routine clinical conditions. During prolonged administration of parenteral nutrition the most important criteria of its efficacy are stability or normalization of the body weight and of the main biochemical findings, such as the concentration of albumin, urea, transaminase, and glucose in the serum. The criterion of efficacy of parenteral nutrition of small children is increasing body length and weight, which become normal for a given age. Assessing efficacy of short-term parenteral nutrition of children in critical conditions is very difficult, the observations are usually limited to a thorough control of the biochemical changes in the blood serum and urine.

## Chapter 27

## Intensive Therapy of Acute Renal Failure

Acute renal failure is characterized by severe renal dysfunction, which is manifested by disordered water-electrolyte metabolism and upset acid-base equilibrium, by impaired withdrawal of protein metabolites (rest nitrogen), by a significant disturbance in the circulation of blood and lymph in the kidneys with subsequent development of azotaemia and uraemia

*Aetiology and pathogenesis* Acute renal failure is a sequel to various pathological processes, by which blood circulation in the kidneys is impaired, or by which a pronounced nephrotoxic effect is imposed. Acute renal failure is a polyaetiological disease, which is always secondary in its character. Acute renal failure can be provoked by traumas associated with blood loss, burns of various degree, exposure to nephrotoxic substances, surgical trauma associated with upset water-electrolyte metabolism, transfusion of incompatible blood (incompatible with respect to the blood group or the Rhesus factor), bacterial invasion with haemolysis, bacteriaemic shock, or allergic reactions

The condition of the patient before the onset of acute renal failure is also very important. This condition depends on the primary disease, from which the patient suffered before the above-mentioned provoking factors promoted the onset of acute renal failure. The primary disease may be associated with upset water-electrolyte metabolism, latent chronic nephropathy, etc. Acute renal failure can develop due to severe cell invasion of the interstitial tissue of the kidney, which often concurs with intense cell decomposition, formation of considerable amounts of uric acid and obstruction of the renal tubules with its crystals

Despite the numerous aetiological factors, the pathogenetic mechanisms of the disease are quite general. At the present time the ischaemic theory is quite popular. Ischaemia is a very important aetiological factor of acute renal failure occurring due to circulatory disorders in shock of various aetiology. It develops as a result of spasm of the renal vessels, the afferent vessels of the glomeruli included, thus arresting glomerular filtration. This occurs in the presence of general hypotonia with a fall of arterial pressure below 60 mm Hg. But the fall of the arterial pressure is not the main causative factor, because acute renal failure often occurs in the presence of normal blood pressure. This indicates involvement of more complicated regulatory renal mechanisms. The onset of acute renal failure is connected with affection of the renal tubules due to anoxia of the renal tissue

When the body is exposed to a nephrotoxic substance, acute

renal failure occurs not only due to poisoning of the kidneys but also due to disturbed renal blood flow. Degenerative and necrotic changes occur in the epithelium of the proximal tubules. These are diffuse in their character. In other words, the changes occur at sites where the toxic substance is reabsorbed after it has passed the glomerular filter. The intercurrent ischaemia completes the set of pathological changes and causes the breakdown of the basal membrane. Anuria can develop as a result of considerable affection of the renal parenchyma, compression of the ureters by tumour, or obstruction of their patency. Anuria can also be caused by unilateral occlusion of the ureter and the reflex depression of the other kidney.

Liver dysfunction is also an important pathogenetic factor of acute renal insufficiency. The liver and the kidneys have very intimate ontophylogenetic and physiological connections. Moreover, during shock these two organs are equally affected by ischaemia. The condition can be characterized as the hepatorenal syndrome. The clinical picture of such cases is characterized by the prevalence of signs of liver affections. Lethal outcome is possible. Considerable dystrophic and necrotic changes can be seen in the liver.

*Clinical picture* Four stages are distinguished in the clinical picture of acute renal failure: 1—the initial stage (shock), 2—oliguria and anuria, 3—restoration of diuresis, and 4—recovery. All these stages can be distinguished only if the disease runs a benign course. If the condition of the patient worsens, the second stage converts into uraemic coma. During the early stage the symptoms of acute renal failure are often attended by signs of shock. Depending on the cause of renal failure, either symptoms of renal affections or shock will prevail. For example, pronounced signs of shock and local symptoms prevail in patients with trauma (the syndrome of prolonged compression). In poisoning the signs of gastrointestinal dysfunction and shock will prevail. The symptom, which is common for both cases, is circulatory collapse, which can be only transient and remain unnoticed, while in other cases it can last for several days. Glomerular filtration can be disturbed during this stage due to the insufficiency of renal blood circulation. Diuresis decreases to 300-500 ml a day and anuria occurs in some cases. The clinical signs of renal failure of the first stage are not pronounced and can remain unnoticed because of the gravity of the main disease and shock. The course of the first stage runs simultaneously with the clinical picture of general circulatory disorders and lasts for 1 or 2 days.

The symptoms of renal affections prevail during the second stage of acute renal failure. The clinical picture is characterized by oliguria (the daily diuresis decreases to 300 ml in older children and to 50-60 ml in nurslings) or by anuria (the diuresis decreases to 50 ml in older children and to 10 ml in nurslings). The urine of children

with oliguria is dark and turbid. The specific gravity of the urine is low (1.009-1.010). Microscopy of the sediment reveals the presence of great number of erythrocytes, leucocytes, epithelial cells, and bacteria. Haemoglobin grains are found in intravascular haemolysis. The condition of patients during this stage is critical. Adynamia develops. The body temperature is either normal or elevated. As the reactivity of the child decreases, the temperature rise can be insignificant. The skin is dry and scaling. The nervous system is affected, which is characterized by asthenia, headache, increased fatigue, muscular excitation, convulsions, psychic disorders with acute delirium and suicidal attempts.

Upset water-electrolyte balance is an important factor of the clinical picture in acute renal failure. Urinalysis reveals proteins, increased quantity of leucocytes, erythrocytes, and casts. The blood picture is characterized by hyponatraemia (to 120-125 mmole/l), hyperkalaemia (to 5-7 mmole/l), hypocalcaemia (1.5-2 mmole/l), and hypermagnesaemia (1-1.5 mmole/l). The chlorine concentration in the blood decreases to 85-95 mmole/l, while the concentration of phosphorus increases to 2.4-5 mmole/l. The concentration of organic acids increases causing acidosis. The alkaline reserve of blood diminishes. Hypoproteinaemia (60 g/l, or less than 3 g%) and dysproteinaemia (caused by increased content of large protein fractions) arise along with a considerably increasing level of unbound nitrogen (57 mmole/l, or 80 mg%).

Derangement of water excretion causes disturbances in the water metabolism due to intracellular or extracellular hyperhydration. Intracellular hyperhydration is characterized by affections of the alimentary tract and the nervous system (vomiting, anorexia, headache, convulsions, and coma), while the extracellular hyperhydration is attended with oedema and signs of cardiovascular insufficiency.

The blood picture during the second stage of acute renal failure is characterized by the progress of hypochromic anaemia with a markedly decreased erythrocyte count (to  $1 \times 10^6 \text{ l}^{-1}$  and less) and decreased haemoglobin (to 2.78-5.42 mmole/l, or 4-6 g%). Leucocytosis is also significant to  $15-30 \times 10^9 \text{ l}^{-1}$  with prevalence of neutrophils. Leucocytosis develops in response to the infectious factor or progressive azotaemia.

The third stage of acute renal failure (restoration of diuresis) is characterized by a gradually increasing volume of daily diuresis. The amount of excreted urine during this period can be large, and the third stage is therefore sometimes called polyuric. But the specific gravity of this urine remains low (1.010-1.012) and it contains great amounts of protein, erythrocytes, leucocytes, and bacteria. The amount of excreted nitrogen is low (less than 1 g). Increased amounts of products of nitrogenous metabolism are sometimes found

in the blood, which can be explained by insufficient nitrogen excretory function of the kidneys. The glomerular filtration is only restored during the third stage, while the tubular filtration remains deranged. The electrolyte metabolic disorders are also significant. Polyuria can involve hypokalaemia, hypomagnesaemia and dehydration, which can cause death. During this stage of the disease the children are flaccid, asthenic and dramatically inhibited. Vomiting is copious. Dehydration causes wasting. Coma is possible.

**Diagnosis** The diagnosis of acute renal failure is based on the anamnestic and clinical findings. It is necessary to know when oliguria and anuria developed. Laboratory examinations of the renal functions are of great diagnostic importance. The analysis of blood and urine (in addition to the general analysis) should also include the determination of the volume of circulating blood, haematocrit, the concentration of the electrolytes (in the plasma, erythrocytes, and the urine), endogenic creatinine clearance (in the blood and urine), reabsorption of water, concentration of amine nitrogen, rest nitrogen, total protein and its fractions, the Zimnitsky test should also be carried out. Table 26 gives some normal indices in children.

Table 26 Indices of Normal Renal Function in Children (according to Veltishchev et al, 1970)

| Test                | Medium       | Age            | Quantity                  |
|---------------------|--------------|----------------|---------------------------|
| Amino acid nitrogen | Plasma       | Newborns       | 5 35-6 78 mmole/l         |
| Rest nitrogen       | Plasma       | Older children | 3 21-5 35 mmole/l         |
| Free ammonia        | Blood        | Newborns       | 35 7-39 3 mmole/l         |
| Total protein       | Plasma       | Older children | 14 3-28 6 mmole/l         |
| Haematocrit         | Blood        | All ages       | 7 1-14 $3 \times 10^{-3}$ |
| Potassium           | Plasma       | Newborns       | 46-69 g/l                 |
|                     |              | Older children | 59-81 g/l                 |
|                     |              | Newborns       | 0 42-0 54                 |
|                     |              | Older children | 0 35-0 48                 |
|                     |              | Newborns       | 4 5-6 5 mmole/l           |
|                     |              | Older children | 3 6-5 5 mmole/l           |
| Sodium              | Erythrocytes | 1-14 years     | 93 6-115 8 mmole/l        |
| Creatinine          | Plasma       | All ages       | 137-152 mmole/l           |
|                     | Plasma       | All ages       | 354-106 $\mu$ mole/l      |
|                     | Blood        | All ages       | 44 2-177 $\mu$ mole/l     |
| Water reabsorption  |              | All ages       | 97-99 per cent            |

In most cases it is not difficult to establish the cause of acute renal failure because it develops next to aggression. The difficulties arise in the absence of anamnesis and if the child's condition is critical. The diagnosis becomes difficult to establish when there is a combination of factors each of which can cause acute renal fail-



ure independently, while information on the child's renal function prior to the onset of acute failure is absent

It is very important to establish the cause of onset of acute renal failure because further condition of the child and prognosis largely depend on the timely taken correct measures. For example, if transfusion of incompatible blood is the provoking factor, it is necessary to carry out exchange transfusion as soon as possible.

It is often difficult to establish the cause of acute renal failure during the post-operative period. It can be due to disordered water-electrolyte metabolism, transfusion of incompatible blood, non-replenished blood loss, or due to exacerbation of latent pre-operative nephropathy. Complications of the main disease during the post-operative period (peritonitis, dynamic intestinal obstruction, incoercible vomiting, etc.) can also be the cause of acute renal failure.

In cases with anuria catheterization of the ureters is indicated if their occlusion is suspected. Retrograde pyelography and scout picture of urinary ducts should also be done. Catheterization of the ureters is contraindicated if the cause of acute renal failure is known.

A thoroughly collected anamnesis facilitates differentiation between acute renal failure and other forms of renal insufficiency in urological diseases. The physician may encounter with cases where several factors can be differentiated and each of which can cause acute renal failure. Only timely and correct diagnosis can suggest the correct therapy in such cases.

The absence of oliguria makes the diagnosis of acute renal failure difficult. For example, in cases with severe burns, diuresis can remain almost normal, but it does not indicate the absence of renal insufficiency. Low specific gravity of the urine is an indication of developing acute renal failure, which can be timely diagnosed by measuring the daily diuresis and determining rest nitrogen and the specific gravity of the urine. Determining hourly diuresis is also important diagnostically. If 20-40 ml of the urine are excreted within an hour, shock kidney is absent.

It is difficult to diagnose acute renal failure developing in the presence of latent nephropathy. The disease is always exacerbated on exposure to cold, in infections, and faulty diet. A correctly collected anamnesis helps establish differential diagnosis. Urological and bacteriological examination of the urine, and urinalysis according to Addis-Kakovsky-Hamburger help diagnose chronic nephropathy. Renal failure in such children is especially acute and rapidly progressing. Uraemia increases without decreasing diuresis, with symptoms of hypo- and isosthenuria. Azotaemia increases slower than in acute renal failure. The following changes in the electrolytes are observed with the same azotaemia level in the presence of a chronic renal disease: hypocalcaemia is more pronounced, the potassium content is slightly increased or normal, hyponatraemia, as-

sociated with the loss of great amounts of sodium, and also with inability of the kidneys to substitute the hydrogen and ammonia ions for sodium, develops. Anaemia develops in chronic renal insufficiency faster than in acute failure.

*Intensive therapy.* Therapy of acute renal failure should be complex and must include both renal and extrarenal methods of clearance. Conservative therapy is indicated for oliguria (without affection of the central nervous system). The therapy is usually aimed at eliminating the aetiological and pathogenetic factors that provoke acute renal failure. The therapy should be administered at the first stage of the disease.

The therapy begins with management of shock and haemodynamic disorders and includes the following:

(a) replenishment of blood loss by infusion of blood, plasma and plasma substitutes,

(b) using pressor amines, e.g. norepinephrine, epinephrine. The preparations are administered for hypotension, but only after circulating blood volume is normalized, the dose is 0.1 ml per year of age, the preparations have a favourable effect on renal blood flow,

(c) vascular collapse should be treated with adrenal cortex hormones, e.g. hydrocortisone, in a dose of 3-5 mg/kg or prednisolone, 1-2 mg/kg, these doses can be increased 3-4 times in especially severe cases,

(d) cardiac glycosides should be given for circulatory disorders due to cardiac pathology (strophanthin, corglycon),

(e) aminophylline-type preparations (2.4 per cent solution intravenously, 1 ml per year of age), analgesics and antihistaminics should be administered for disordered peripheral blood circulation associated with spasms of renal vessels, e.g. high systolic and low pulse pressure, cyanosis of the mucosa and terminal phalanges, etc., it is desirable that the arterial pressure does not fall below 60 mm Hg,

(f) anaesthesia is important in renal failure following severe traumas, prolonged compression syndrome, narcotic analgesics (e.g. promedol, omnopon) should be used (0.1 ml of a 1 per cent solution per year of age),

(g) antidotes should be administered in poisoning during the first stage of acute renal failure, unithiol should be given to patients poisoned with salts of heavy metals (1 ml per 10 kg body weight).

The therapy of the second stage of acute renal failure should be directed at eliminating the cause of the syndrome and correcting haemostasis (azotaemia, hypotonia of the plasma, acidosis, and water-electrolyte disbalance).

1. The most important step is correction of the water-electrolyte metabolism. The loss of potassium, sodium, magnesium, and chlorine, and also their concentrations in the plasma and erythrocytes,

should be thoroughly controlled. It is also very important to control the ratio between the uptaken water and water lost from the body. All water lost through the kidneys, the skin, the lungs, and the gastrointestinal tract should be taken into consideration. When the lost liquid is replenished, it is necessary to maintain a slight hypohydration in the body. Lack of water rapidly causes azotaemia, while excess liquid results in hyperhydration. The general rule is to give the patient about the same amount of water that was lost during the previous day.

The water balance should be controlled by (a) daily weighing of the patient, (b) measuring uptaken and excreted liquid, (c) determining the haematocrit, haemoglobin concentration, the volume of circulating blood, and total protein.

If the patient is conscious and vomiting is absent, about 60-70 per cent of liquid is taken per os. Otherwise liquid should be administered parenterally.

Correct diet of adequate caloric value (carbohydrates and fats) is very important in the therapy of acute renal failure. The diet should also include proteins, which should have high biological potency (egg protein).

Anabolic hormones, e.g. methandrostenolon (nerobol), or retabolil should be administered to decrease catabolism.

If the child can eat, his diet should not contain foods rich in potassium (milk, fruits, raisins, lemon, oranges, juices, etc.). In the presence of anuria potassium or sodium preparations should be absolutely excluded. Blood taken not later than two days ago should be transfused. The danger of hyperkalaemia increases in the presence of hypocalcaemia and marked acidosis. In hyperkalaemia it is necessary to administer large volumes of hypertonic calcium chloride and calcium gluconate solutions, and also hypertonic glucose solution with insulin, which promotes formation of glycogen with consumption of potassium. Extracellular potassium thus passes into the intracellular one.

2. Acidosis should be controlled by administering a 4 per cent sodium bicarbonate solution (the dose depending on the acid-base balance).

3. Diuretics are indicated to increase diuresis in the second stage of acute renal failure. The best effect is attained with the saluretic furosemide (lasix), which should be administered in a dose of 3-5 mg per kg body weight a day. Good results are attained with the osmotic diuretic mannitol, which is indicated for oedema. In the absence of oedema mannitol can only be administered after transfusion of at least 100-150 ml of liquid. A 15-30 per cent mannitol solution is used for the purpose.

4. Bacterial flora activates, if the body reactivity decreases. Antibiotics should therefore be administered in acute renal failure.

to suppress bacterial flora. It should however be remembered that antibiotics have nephro- or hepatotoxic properties. Antibiotics of the penicillin series should therefore be preferred. The doses can be large (to 300 000-500 000 units per kg body weight). Nystatin is obligatory for use.

Daily control of the electrolyte level, diuresis, total protein, haematocrit, haemoglobin, rest nitrogen, urea, blood creatinine, and acid-base balance is used to assess efficacy of the therapy administered. If the therapy is effective in the oliguria stage, development of severe renal failure can be prevented. If the therapy fails to give the desired effect, clearance should be ensured by extrarenal means.

During the third stage of acute renal failure (restoration of diuresis) it is necessary to ensure the administration of large volumes of liquid, since otherwise the patient may develop significant dehydration and die.

1 The most important item is correction of water-electrolyte metabolism (control of hypokalaemia and hyponatraemia). The amount of potassium and sodium to be administered should be calculated with reference to the volume of the extracellular space. The amount of the extracellular fluid is on the average 20 per cent of the body weight (this figure is significantly greater in infants). For example, the potassium content of blood plasma in a 10-year-old child weighing 30 kg is 3 mmole/l. The amount of the extracellular fluid in the child is

$$\frac{20 \times 30}{100} = 6 \text{ litres}$$

The potassium deficit is  $50 - 30 = 20$  mmole/l. One mmole/l of potassium is contained in 1 ml of a 7.5 per cent potassium chloride solution ( $1 \text{ mmole/l K} = 74.5 \text{ mg KCl}$ ). In our case the child should be administered 12 ml of a 7.5 per cent potassium chloride solution. Potassium should be administered slowly by drip infusion of a solution of glucose with insulin in the concentration of 0.2-0.3 per cent.

The needed amount of sodium should be determined in a similar way. It should only be remembered that 1 mmole of Na is equivalent to 59 mg of NaCl or 84 mg of  $\text{NaHCO}_3$ . When replenishing the sodium loss, it is not recommended to use only the isotonic sodium chloride solution because much chlorine is administered with the solution, while the kidneys are incapable of managing hyperchloraemia.

The water-electrolyte metabolism should be corrected with constant biochemical control of the blood.

2 As diuresis is re-established, parenteral administration of liquid should be gradually decreased, while the liquid intake per os should be increased. Fruits and juices should be given in increased amounts.



(b) oedema of the lungs or the brain, (c) hyperkalaemia (above 7 mmole/l), (d) increased urea content (over 5 mmole/l), (e) decreased alkaline reserve (below 12 mmole/l  $\text{CO}_2$ ), (f) psychic disorders, (g) uraemic coma.

*Contraindications* for haemodialysis are (a) disordered cerebral circulation, and (b) acute myocarditis

In acute renal failure haemodialysis should be used only in cases where other measures proved ineffective. Extracorporeal haemodialysis can be conducted repeatedly at 1-3 day intervals

## Chapter 28

### Intensive Therapy of Acute Hepatic Insufficiency

Acute hepatic insufficiency is a rapidly progressing severe condition with pronounced impairment of all hepatic functions and acute necrosis of the liver cells (hepatocytes) characterized by some specific symptoms

*Aetiology* The causes of acute hepatic insufficiency are quite varied. They are divided into six major groups

1 Acute and chronic hepatitis, cirrhosis, alveococcosis, and tumours of the liver (both primary and metastatic)

2 Upset intrahepatic blood circulation associated with occlusion of the liver veins (Budd-Chiari syndrome)

3 Diseases aggravated by developing extrahepatic cholestasis, cholelithiasis, tumour of the common hepatic or common bile duct, tumour or stenosis of the duodenal (Vater's) papilla, chronic pancreatitis with obturation of the common bile duct, and affections of the biliary ducts

4 Diseases of other organs and systems, such as systemic diseases of the connective tissue (collagen disease), endocrine disorders, cardiovascular and infectious diseases

5 Poisoning with hepatotoxic substances, such as phosphorus, carbon tetrachloride, lead, chloroform, halothane, some plants and mushrooms, intolerance of medicines

6 Various injuries, such as burns, vast operative and other traumas with formation of large wounds, massive blood loss, severe purulent complications, blood transfusion, and also septic abortion

Among the causes of acute hepatic insufficiency in children the leading role is indisputably attributed to viral hepatitis (in about 70 per cent of cases)

*Pathogenesis* The pathogenesis of acute hepatic failure always depends on the affection of cell microstructures—hepatocyte organelles

Table 27 Symptoms of Hepatic Insufficiency and Coma

| Degree of gravity                                  | Psychic condition   | Tremor                           | EKG changes               |
|--|---|----------------------------------|---------------------------|
| <i>Degree I</i>                                    |   |                                  |                           |
| Prodromal changes (often revealed retrospectively) | Euphoria, sometimes depression, dimmed consciousness Inhibition Absence of orientation                              | Frequent but can be absent       | Usually absent            |
| <i>Degree II</i>                                   |   |                                  |                           |
| Precoma  | Dimmed consciousness Euphoria and sleepiness are common Behavioural disturbances                                    | Frequent and quite obvious       | Occur in almost all cases |
| <i>Degree III</i>                                  |   |                                  |                           |
| Stupor   | Almost continuous sleep, but the patient can be waken up Marked dimness of consciousness and absence of orientation | Usually present                  | Occur in almost all cases |
| <i>Degree IV</i>                                   |   |                                  |                           |
| Subcomatose condition or coma                      | Complete absence of consciousness Reaction to pain may be present or absent, depending on depth of coma             | Usually absent (muscular atonia) | Present                   |

Damage to ribosomes and endoplasmatic network upsets the protein synthesis Mitochondrial affection involves disturbances in the processes occurring with energy consumption, in the first instance-catabolism and synthesis of neutral fats, phospholipids Many biochemical reactions, by which toxic metabolites are detoxicated, occur in the organelles The main pathogenetic mechanisms in development of acute hepatic failure, which are responsible for the varied clinical manifestations, are disturbances in protein synthesis and gradually increasing level of active endogenic toxins in the blood For example the prime cause of haemorrhagic syndrome is upset synthesis of II, V, VII, and X blood coagulation factors, which are synthesized exclusively by the liver cells

Gradual accumulation of free ammonia in the blood causes metabolic alkalosis and hence hypokalaemia and hyponatraemia Penetration of ammonia through the blood-brain barrier is much facilitated in alkalosis This causes nervous and psychic disorders (up to coma)

*Clinical picture and diagnosis* The characteristic clinical symptoms are dyspepsia (nausea, vomiting, anorexia), increasing haemorrhagic syndrome (petechiae, nasal and gum bleeding), icteric skin and sclera, decreased tissue turgor, and specific hepatic breath Splenomegaly, ascites, nervous and psychic disturbances are not infrequent Psychic and nervous disorders are so characteristic of hepatic insufficiency that the latter was classified in accordance with severity of psychic and nervous disturbances (see Tables 27 and 28)

The comparison of severity of hepatic insufficiency with biochemical changes occurring in this pathology is given in Table 28

*Intensive therapy* The therapy of hepatic coma in children includes the following

Table 28 Hepatic Function in Children

|                        | Degree I (mild) | Degree II (medium)<br>gravity) | Degree III (severe)                 |
|------------------------|-----------------|--------------------------------|-------------------------------------|
| General condition      | Satisfactory    | Medium gravity                 | Severe                              |
| Skin colour            | Ordinary        | Icteric sclera                 | Icteric skin and mucosa             |
| Liver                  | Normal          | Increased by 1-2 cm, painless  | Increased by more than 2 cm, tender |
| Bilirubin (total)      | 21-26 mmole/l   | 26-36 mmole/l                  | 36 mmole/l                          |
| Cholesterol (total)    | 3 1-3 4 mmole/l | 2 6-3 1 mmole/l                | Less than 2 6 mmole/l               |
| Lipids (total)         | 4-8 g/l         | 3-4 g/l                        | Less than 3 g/l                     |
| Protein (total)        | 65-68 g/l       | 60-65 g/l                      | Less than 60 g/l                    |
| Bromsulphthalein test  | 5-10 per cent   | 10-20 per cent                 | More than 20 per cent               |
| Albumin-globulin ratio | 1 10-1 30       | 0 90-1 10                      | Less than 0 90                      |



1 Complete abstention from protein intake, restricted intake of salt in the presence of oedema or ascites, and complete suspension of methionine and choline administration in connection with high azotaemia

2 Parenteral nutrition Hypertonic glucose solutions should be administered intravenously. A 10 or 20 per cent glucose solution should be used (120-150 ml per kg body weight). Insulin (1 unit per 1 g of dry glucose) should be added to glucose

3 Intravenous administration of glutamic acid (calcium or magnesium glutamate). A 10 per cent solution is used in a dose from 2 to 10 ml, daily or every other day, in a course of 20 injections. A 10 per cent calcium chloride is also administered in a dose of 1 ml per year of child age

4 In order to decrease protein absorption in the intestine, laxatives should be used, e.g. magnesium sulphate. Antibiotics of broad-spectrum (neomycin, monomycin, and others) are given for the same purpose

5 Corticosteroids should be administered intravenously or intramuscularly in large doses. Their effect is based on the anti-inflammatory and anti-allergic properties, and also on their ability to decrease the formation of fibrous tissue and intrahepatic cholestasis. Prednisolone is the most effective preparation. Its doses should be increased significantly (to 10 mg/kg) in coma

6 Frequent inhalations of oxygen (and also its administration per os) prevent hypoxia and improve oxygenation. Enteral administration of oxygen should be ensured through a gastric tube. Hyperbaric oxygenation is also effective. Arteriovenous shunting is now used. To that end, the portal vein of the liver is catheterized through the umbilical vein, with subsequent communication of the portal vein with the radial artery (through a polythene catheter)

7 A better withdrawal of rest nitrogen is attained through (a) exchange blood transfusion, (b) cross circulation with a donor, (c) cross circulation with hog liver. But a stable improvement can only be attained with frequent repetition of the procedure during several days till the own hepatic function of the patient is normalized. The efficacy of the last method (c) is however disputable. The effect of animal liver on the child's liver during extracorporeal circulation of blood should be further studied in more detail

8 Haemodialysis and haemoperfusion should be used in severe hepatic failure

9 Drainage of the thoracic lymph duct is effective

A world wide search for efficacious pathogenetic and symptomatic methods of treating hepatic insufficiency is now being undertaken. But only a thorough study of their effect on the child's liver can prove their actual efficacy

Hepatic insufficiency (other than coma) can also be treated by the following complex therapy

1 Sufficiently caloric diet (1800-2000 kcal/day) fasting intensifies catabolism, which increases in children with hepatic insufficiency. Catabolic processes are depressed by anabolic hormones given in a dose from 2 to 5 mg a day. The diet should be rich in protein and carbohydrates with restricted (or fully absent) fats. But some authors doubt if it is actually necessary to restrict or exclude fats from the diet. In their opinion the amounts of proteins, fats and carbohydrates in the diet should relate as 1:1:5.

2 Administration of vitamins, especially vitamins B, and also vitamins C, A and K in large quantities. Vitamins B<sub>12</sub> and B<sub>15</sub> (in doses calculated with respect to age) are indicated. Vitamin B<sub>12</sub> has lipotropic properties. Vitamin B<sub>15</sub> is indicated for its effective protection of the liver when it is exposed to various noxious factors, especially hypoxic and toxic ones. Vitamin B<sub>15</sub> has also lipotropic and detoxicating properties and improves utilization of oxygen.

3 Blood and plasma transfusions. Blood taken not later than 2 days ago should preferably be transfused. Glucose is also important. Its parenteral administration intensifies glycogenization of the liver. Moreover, glucose donates glucuronic acid, which is necessary for normal detoxicating function of the liver.

4 Administration of lipotropic substances capable of protecting the liver from fat degeneration. They prevent deposition of fat in the liver by phosphorylation. These substances include choline, methionine, lipocaine, choline chloride, etc. The course of treatment with lipotropic substances should not last more than 12-14 days. The doses depend on age. Choline chloride is given in a dose of 1-2 g a day, this dose protects the liver of the child from some fats and inhibits development of cirrhosis. The preparation is administered slowly into the vein in 200 ml of a 5 per cent solution of glucose or isotonic solution. (The rate of infusion should not exceed 15-20 drops per minute.) If the dose is insufficient, or if the substance is administered with intervals, necrotic foci can form in the liver and kidneys.

5 Administration of glutamic acid and glutathione. The effect of glutathione is explained by the presence of the SH groups in its molecule, which promote detoxication processes. Glutathione is administered daily, into the vein, in a dose of 500-1000 mg.

6 Intravenous administration of a 20 per cent albumin solution, blood, plasma, and protein hydrolysate. These preparations should be administered for hypoproteinaemia, which occurs in patients with hepatic insufficiency and in the presence of prevailing amounts of coarse proteins.

7 Intraventricular or intraintestinal administration of oxygen for hypoxia. This increases PO<sub>2</sub> of the portal blood and the oxygen

consumption in the liver Hyperbaric oxygenation seems to be the most effective means to control liver hypoxia

8 Therapy with preparations made of fresh liver extracts (syiepar, campolon, hepalon, etc ) These preparations are free from protein and contain active substances of healthy liver

9 Obligatory use of corticosteroids The administration of prednisolone should be begun with 10-15 mg the dose being then increased to 80-100 mg daily Maintenance doses of prednisolone (5-10 mg) can be given for several months Prolonged courses of corticosteroids should be combined with administration of potassium and in some cases anabolic hormones (nerobol, dianabol) and other preparations improving metabolic processes in the liver and promoting synthesis of proteins

Intraportal infusion of solutions has been used in recent years The portal vein is catheterized by bougienage of the umbilical vein This method is believed to be more effective than intravenous administration, it rivals the efficacy of intra-arterial infusions

10 Maintenance of metabolic processes during operative intervention is necessary to prevent hepatic insufficiency during the post-operative period Local hypothermia of the liver (34-32°C) with simultaneous decrease of the rectal temperature to 34°C can be used to ensure effective protection of the liver and to increase its resistance to hypoxia and other injurious effect During surface hypothermia of the liver the metabolic rates are decreased and the activity of the serum enzymes increases

## Chapter 29

### Intensive Therapy of Acute Exogenous Intoxication

Acute exogenous intoxication (poisoning) stands at the top of list of emergencies in children The abundance of domestic chemicals, strong medicines, and also improper and faulty use of other medicinal preparations have resulted in an abrupt rise in the incidence of poisoning The specific behaviour of a child, which is characterized by high activity, curiosity, the desire to taste unknown substances, a relatively large gulp (1 ml per kg body weight, which is larger than in adults), arrogance of many parents, and improper storage of medicines and chemical poisons have increased the danger of poisoning in children Statistic evidence shows that lethal outcomes from acute poisoning in children in developed countries greatly exceed the mortality rate from all infectious diseases taken together

Poisoning is an important item of health education of parents and

medical workers. It is interesting to note that poisoning occurs more frequently among more active children, which take interest in the surroundings. And it should also be remembered that it is much easier to prevent poisoning than to treat it. A paediatrist should therefore be always aware of possible poisoning. Poisoning is highly probable in children's emergencies of unknown aetiology, especially in cases with signs of affections of the central nervous system.

*Aetiology and epidemiology* The causes of acute poisoning in children are quite varied. They depend on age, the season of the year, residence and many other factors. Poisoning of neonates and nurslings often depends on poisoning of their mothers who take noxious substances or medicines. Exogenous intoxication is thus caused by intake of poisoned mother's milk. Infants ageing under 3 years can be poisoned due to overdosage of medicines. Instillation of atropine under the conjunctiva or during irrigation of the lacrimal ducts is an example of specific poisoning.

Urban children would usually be poisoned with medicines or domestic toxic substances, while children of rural areas are usually affected with plant poisons, such as mushrooms, insecticides, and the like. The incidence of poisoning depends on season. Of course, it is impossible to mention all possible cases of poisoning in children. It is interesting to note that more than 250 different medicinal, plant and chemical substances stand in the list of causes of poisoning in children compiled at the Children's Toxicology Centre of the Moscow Medical Institute.

*Pathogenesis* The mechanism of pathological action of poison is varied too and depends on the type of poison, route of intake and the child's condition. Two main routes, by which poison acts on the body, are distinguished, viz., local and general, associated with absorption of poison into the blood.

When ingested, medicines and corrosive substances such as benzine, acids, or alkalis burn the mucosa of the oropharynx, oesophagus, stomach, and the intestine. When poison attacks the eye conjunctiva, the skin and mucosa, it causes a burn. Gaseous substances (carbon monoxide, benzine vapour) affect the respiratory mucosa and cause pneumonia.

The general resorptive effect is associated with passage of poison into the blood, thus causing harmful effect on the vital bodily functions irrespective of the route by which the poison penetrates the body. As a rule, poison is absorbed through the gastric mucosa. But poison resorption is also possible through the respiratory mucosa and the skin. Cases were reported of severe mercury poisoning of children who were treated by local application of mercury ointments. Poisoning with aniline dyes are also very severe. Cases were reported where children were poisoned by contact with aniline-printed laundry labels.

Depending on their properties, toxic substances undergo complicated conversions in the child's body, they are partly hydrolysed and bound with various components of biological fluids, such as proteins of plasma or fats. The poison first circulates with the blood to disturb the enzyme processes and alter the action of the mediating systems, and is then carried to various tissues. But the action of poison is not restricted to its local and general effect on the child. Poisons often cause reflex reactions to pain (if the poison is a corrosive substance), to irritation of the mucosa of the stomach, etc. Toxic agents can thus cause severe dysfunctions irrespective of the route of their ingress. A local effect, for example, burn of mucosa of the oesophagus and stomach with necrosis of tissues and perforation of the stomach wall, is very dangerous in itself, but it also involves the resorptive action of poison. Sometimes poisoning with medicines or mushrooms has the most dangerous effect on the cardiovascular system, the liver and other organs. Poisonous agents disturb enzymatic systems and interfere with all spheres of metabolism and homeostasis, but they can also cause an indirect effect on the function of the central nervous system, circulation of blood and respiration.

*Clinical picture* The clinical course of acute poisoning depends on the aetiological factors and is therefore quite varied. But in most cases it is characterized by general and specific symptoms. The *general* clinical signs of poisoning in a child are usually manifested by changes in the central nervous system: flaccidity, adynamia, disordered coordination of movements, depression, monotonous and scanning speech, and staggering gait. The skin and tendon reflexes are intensified or depressed. As intoxication progresses, motor and psychic anxiety, hallucinations or, on the contrary, depression of consciousness and comatose condition develop.

Respiration is disordered to acute respiratory insufficiency with retraction of yielding sites of the chest, involvement of the accessory muscles, upset respiratory rhythm, development of pathological respiration and apnoea. All these changes can be caused by the effect of poison on the respiratory centre and the neuromuscular transmission, or enzymatic processes. Hypoxia is no less dangerous. It can be due to derangement of the respiratory function of the blood. Normal respiration is often disturbed by aspiration of the vomitus.

Circulatory disorders are usually manifested by changes in the heart rate and arrhythmia, dullness of heart sounds, decreased arterial pressure, metabolic and hypoxic changes in the myocardium. This complex of symptoms is sometimes called the 'toxic myocardiopathy'.

Gastrointestinal disorders are common for most poisoning cases in children. These are abdominal pain, nausea, vomiting, intestinal paresis, or frequent liquid stools. Dysfunction of the kidneys and

liver is common for severe poisoning. The functional changes later convert into organic.

*Specific* symptoms of poisoning are characteristic for certain cases of poisoning. In severe poisoning these develop in the presence of general changes in children. Below given are the specific symptoms frequently occurring in poisoned children.

1 The specific smell suggests poisoning with kerosene, benzene, or alcohol.

2 Burnt skin and mouth mucosa in cases with ingestion of acids, alkalis, unslaked lime or potassium permanganate.

3 Cyanosis in poisoning with aniline, nitrobenzene, saltpetre, and sodium nitrate.

4 Skin haemorrhage in poisoning with heparine, phenylene, benzene, xylene, or salicylates.

5 Haematuria in poisoning with acetic acid, iodine or potassium chlorate.

6 Convulsions in poisoning with adrenaline, aminazine, their analogues, analgin, butadione, cardiac glycosides, or strychnine.

7 Dilatation of the pupils in poisoning with atropine, codeine, belladonna, trioxazine, or henbane.

8 Contraction of the pupils in poisoning with aminazine, barbiturates and pilocarpine.

9 Hyperhidrosis in poisoning with salicylates or pilocarpine.

10 Elevated body temperature in poisoning with antibiotics, salicylates and sulphur drugs.

*Diagnosis.* Identification of exogenous intoxications in children is based on some factors, such as clinical picture (general and specific symptoms), anamnesis, toxicological situation and biochemical methods of identification of poison in the child.

*Anamnesis* is very important for identification of a particular poison. The parents and other adults, overanxious with the accident, can give incorrect information. The process of collecting anamnesis should therefore be planned. It is necessary to find out what medicines or domestic poisonous substances might be ingested by the child, what was the child's food, if there are similar symptoms in other children at school, kindergarten, or the family, and what changes occurred in the child's condition during recent hours (sleeplessness, vomiting, loss of consciousness). It is necessary to find out if the child drank some unknown liquid, ate some tablets, etc. In addition to these special questions, the adults should be asked the general anamnestic questions about diseases of the past and tolerance of medicines.

Information about the *toxicological situation* should be obtained. This includes inspection of the site where the child was present during possible ingestion of poison, search for vials, ampoules or other containers for medicines and their possible remnants, and also search

for traces of solutions of domestic poisons (acids, alkalis, etc.)

*Biochemical identification* of poisons includes qualitative and quantitative determination of various medicines or other chemical substances in the blood, urine, or other biological fluids of the victim. These examinations should be carried out at special biochemical and toxicological laboratories.

*Intensive therapy* It may appear to someone that it is not necessary to apply 'intensive' therapy to all cases of acute poisoning in children. But it should be remembered that even in mild cases the child's condition may worsen suddenly with increasing signs of intoxication. Therefore, children with mild poisoning, or even only suspected for poisoning, need *intensive* therapy and care. The main methods of intensive therapy of children with acute poisoning are as follows: withdrawal of the child from the enclosure where the poison can still remain, removal of the poison not absorbed in the blood of the victim, and elimination of the poison from the blood (detoxicating therapy, antidote therapy, correction and maintenance of the vital body functions).

*Withdrawal of the child from an enclosure or the site where his exposure to the poison may continue* This first of all concerns evacuation of the child from enclosures where he is exposed to carbon monoxide, as in fire. This also concerns the cases where solutions of corrosive substances, aniline dyes, etc., get on the child's clothes. The child should in such cases be stripped off his clothes and his skin should be washed.

*Removal of poison, which has not yet absorbed in the blood* This should be done as follows. If liquid or powder containing poisonous substances gets on the skin, it should be washed with warm water and soap. The eye mucosa and the conjunctiva should be irrigated with tepid water from a syringe or a rubber bulb. A 1 per cent novocaine solution with adrenaline can then be instilled into the conjunctival sac. The mouth and nasopharyngeal mucosa should also be rinsed with warm water, if the child is conscious. If the victim is unconscious, his mucosa should be treated with a moist gauze ball. Intranasal novocaine block and inhalation of novocaine, suprastin and hydrocortisone should then be administered.

If the poison was ingested, gastric lavage is obligatory. The stomach should be irrigated as soon as possible, at any stage of treatment. Previous vomiting or eating are not contraindications for gastric lavage. Irrigations should be performed through a gastric tube. The trachea of neonates and infants, and also of unconscious victims, should be intubated before gastric lavage. Drinking water (35-36°C) should be used for irrigation. Irrigation should be continued till the washings are clear. Volumes of liquid that can be administered at a time are given in Table 29. It is useful to add a tablespoonful of common salt per litre of water. This solution causes

spasm of the pylorus and prevents further progress of poison from the stomach into the intestine. Salt is contraindicated in poisoning with corrosive substances. If the child is poisoned by large masses of vegetables, mushrooms, or tablets, the irrigation of the stomach should be repeated at 20-30 minute intervals.

Table 29 Quantity of Water for Single Administration During Irrigation of Stomach

| Age     | Volume, ml | Age     | Volume, ml |
|---------|------------|---------|------------|
| Neonate | 15-20      | 2-3 y   | 200-250    |
| 1 m     | 40-50      | 4-5 y   | 300-350    |
| 3-4 m   | 60-90      | 6-7 y   | 350-400    |
| 5-6 m   | 100-110    | 8-11 y  | 400-450    |
| 7-8 m   | 110-120    | 12-15 y | 450-500    |
| 9-12 m  | 150-200    |         |            |

Ingested poisons should be removed from the intestine in all cases. Laxative salts and cleansing enemas are used for the purpose. Vaseline oil or glycerol (2-3 g/kg) are used as laxatives in poisoning with corrosive substances.

*Removal of poison from the blood* is performed by various methods depending on the degree of intoxication. Forced diuresis is used in most cases. Liquid is taken per os (water load) and intravenously. The daily and hourly volumes of liquids administered depend on the degree of exogenous intoxication.

1 In cases with mild intoxication (the child is conscious) the liquid is administered in a dose of 3-5 ml per kg body weight per hour, the rate of infusion can be increased to 6-7 ml/kg per hour.

2 In severe cases the rate of liquid administration is 5-7 ml/kg per hour, and it increases gradually (within 2-3 hours) to 12-15 ml/kg, if diuresis is normal and the cardiovascular function is not impaired, the rate of administration should in two hours be increased to 20 ml/kg per hour.

3 Water is given to drink in slight poisoning. Older children are given tea or other drinks in volumes specified in item 1. Neonates and infants should be given solutions through a gastric tube (fixed to the skin of the face).

4 Forced diuresis is induced intravenously in children with medium gravity and severe poisoning, peripheral or central veins are used for the purpose.

5 The composition of solutions administered varies depending on the child's condition. The simplest composition is 5 per cent glucose ( $1/2$  of the total volume), Ringer's solution ( $1/4$  of the total



volume), and isotonic sodium chloride solution ( $\frac{1}{4}$  of the total volume) In order to shift the condition to the alkalosis side, it is recommended to add 4 per cent of sodium bicarbonate solution (10 per cent of the total administered volume), haemodes (20-40 ml/kg a day), 5-15 per cent albumin solution (10-20 ml/kg a day), and rheopolyglucin These substances are added at the expense of decreasing amount of the Ringer solution and the isotonic sodium chloride solution

6 The child's condition is controlled during forced diuresis by determining the quantity of the urine excreted, the haemoglobin concentration, the haematocrit, the central venous pressure, the electrolyte balance, and the acid-base balance If the amount of liquid excreted by the child within an hour is less than 75 per cent of the administered liquid, diuretics (lasix, mannitol, urea) should be administered

7 The duration of forced diuresis depends on its efficacy If intoxication decreases, the amount of the administered liquid should be decreased If forced diuresis fails to give appreciable effect within 1-2 days and the child remains in the state of coma without positive changes in his condition, more effective detoxication measures should be taken replacement transfusion of blood, haemoperfusion, peritoneal dialysis, and haemodialysis

*Hyperbaric oxygenation* can only conventionally be considered as a means promoting withdrawal of poison from the blood The mechanism of decreasing toxic effect of poison by hyperbaric oxygenation is probably explained by intensified oxidation processes and accelerated hydrolysis of toxic substances Our experience shows that hyperbaric oxygenation is a pathogenetic method of treating children poisoned with carbon monoxide This method is also very effective in poisoning with some other substances, e g nitrites

*Antidote therapy* The principle of the antidote therapy is based on the adsorption of toxic substances on the substances known as antidotes, by which non-toxic complexes are formed, or the antidote may act as an antagonist to the poison Sometimes antidotes act as physiological antagonists and substances, which compete with poisons for the receptors Table 30 gives antidotes to common poisons, by which children are affected most frequently

*Correction of vital functions* The correction of the vital functions, such as the function of the central nervous and cardiovascular systems, respiratory, hepatic, and renal functions, does not differ in principle from their correction and maintenance in other diseases It should be remembered that there is a danger of aspiration of vomitus, of shifts in the water-salt metabolism, and other metabolic disorders

Exogenous poisoning in children often occurs in the presence of respiratory, viral, and other diseases In addition to the specific

Table 30 Antidotes Used in Poisoning of Children

| Poison   | Antidote  |
|--|---|
| Hydrocyanic acid   | Amyl nitrite, 1-2 drops on cotton wool to inhale                                  |
| Hydrogen cyanide, organic phosphorus compounds   | Aminophenol, 3 mg/kg  |
| Acetylcholine, carbocholine, cardiac glycosides, pilocarpine, organic phosphorus compounds | Atropine, 1 mg, repeated administrations till signs of atropinization are obvious |
| Barbiturates   | Bemegride, 3-5 mg/kg  |
| Iron preparations  | Deferoxamin, 15 mg/kg per hr Daily dose, 80 mg/kg                                 |
| Salts of heavy metals  | Dimercaprol (unithiol), 3 mg/kg   |
| Arsenic, lead, mercury   | Sodium thiosulphate, 30% sln, 25 mg/kg  |
| Heparin  | Protamine sulphate, 1 mg neutralizes 1 mg of heparin                              |
| Methyl alcohol, ethylene glycol, butan-phenolic adhesive                                   | Ethyl alcohol, 5% sln, 1 ml/kg  |
| Cardiac glycosides   | Panangin, 0.15-0.3 mg/kg  |

treatment, these patients should also receive treatment for the concurrent diseases. Vitamins are indicated for most cases of poisoning in children.

### Intensive Therapy of Children Bitten by Venomous Animals and Insects

Bites of venomous animals and insects can cause severe exogenous intoxication.

**Snake bites.** Venom of the snake can cause local and general effect on the child. Children in general react more severely to snake bites than adults because the relative dose of the venom in them is greater (6 mg/kg). As a rule, the child feels a strong pain at the site of bite. Hyperaemia can appear at the site. This is followed by cyanosis, oedema of soft tissues and lymphangitis. As intoxication progresses, the general symptoms of poisoning become manifest: nausea, vomiting, thirst, tachycardia, fall of arterial pressure, confused consciousness, delirium, and pyrexia. Severe anaemia may follow. Haemorrhage at the spot of bite spreads by the course of the vessels to reach the internal organs. Muscular debility develops simultaneously. It ends in paralysis of the skeletal muscles, up to apnoea.

**First aid.** The part of the skin bitten by a snake should immediately be seized firmly into a fold and the venom expressed from the wound. The venom can also be sucked off by the mouth. If a resuscitation

tator has wounds in his mouth, he should abstain from this measure. If more than ten minutes have passed after the accident, suction is useless, because the venom has already absorbed in the blood. The bitten extremity should then be immobilized, because the snake venom is carried with lymph, while the lymph flow is slow in an immobile extremity. The child should be soothed; he should be given sedatives, analgesics and much warm water to drink. The child should be transported in the lying position.

Placing tourniquets or burning the bitten site is harmful because these manipulations will add harm to the tissues of the extremity and impair the child's condition, while absorption of the venom will not be prevented.

*Intensive therapy* should be aimed at neutralizing the venom, withdrawing it from the body, and maintaining the bodily vital functions. The Soviet preparation Anti-gyurza (from the name 'gyurza', the venomous snake inhabiting the Soviet Central Asia) is used in a dose of 500-1000 (AU) (antitoxic units) for mild degree of intoxication, 1500 AU for medium, and 2000-2500 AU for severe intoxication. The serum is administered subcutaneously or intramuscularly. The first dose is 0.1 ml, the second dose is injected in 10 minutes (0.25 ml). In the absence of effect, the rest should be injected. Forced diuresis with alkalization of the plasma should be provoked simultaneously. The administered solutions should contain sufficient amounts of vitamins B<sub>1</sub>, B<sub>6</sub>, B<sub>12</sub>, vikasol, ascorbic acid, and hormones. Procaine hydrochloride block (immediately after the bite) or paranephric block are very useful.

In addition to the mentioned treatment, the child should also be given intensive therapy aimed at correcting and maintaining the vital functions.

**Insect bites.** Bites of spiders, latroedectus, tarantulas, and scorpions are especially dangerous. Their venom spreads by the lymph system, passing the blood-brain barrier and affecting the central nervous system to disturb the dynamics and rheology of the blood circulation.

The child feels strong pain at the bitten site, which quickly spreads over the entire body. Headache, dizziness, nausea, and asphyxia develop. As the intoxication progresses, cyanosis, tachycardia and arrhythmia occur. This type of poisoning is characterized by dilatation of the pupils and hypersalivation.

*Intensive therapy* includes immobilization of the child and applying cold to the bitten site. Special serum (antilatrodectus) is administered intramuscularly and in severe cases intravenously (20-50 ml). Hormones, antihistaminics and vitamins are also given. Infusion therapy and correction of circulatory and respiratory function should also be done.

## Chapter 30

## Intensive Detoxicating Therapy

Intensive methods of detoxication are used in sepsis, poisoning and other severe intoxications, when commonly used methods fail

**Haemoperfusion.** By this method blood is passed through special sorbents which are materials with well developed internal structure. The size of their pores is commensurable with the size of the molecules to be adsorbed. Silicon and carbon sorbents have the low selectivity and can retain various substances contained in the blood or lymph. There are also ion-exchange materials, in which the anion (cation) is exchanged for the cation (anion) of the noxious substance contained in the blood or lymph. These are ion-exchange resins of high selectivity.

Haemoperfusion (Fig 57) is indicated for cases where there exists the necessity to remove exogenous or endogenous poisons that pass with difficulty biological membranes (kidneys, lungs) or artificial membranes (artificial kidney), and also poisons that should be eliminated as soon as possible. The conditions that require haemoperfusion include poisoning with preparations whose molecules are large or bound with proteins, combined metabolic disorders, and allergic and autoallergic conditions.

The procedure is not complicated: two large veins (or a vein and an artery) are opened. The patient is given a heparin injection. Blood from the vein (artery) is delivered into a column packed with an adsorbent where the toxic agent is removed from the blood. Purified blood is returned into the circulatory system through the other vein.

At least two circulating volumes of blood should thus be treated.

**Lymphoperfusion.** Purifying lymph by passing it through a sorbing material is called lymphoperfusion. The operating principle is the same as in haemoperfusion.

As distinct from haemoperfusion, *indications* for lymphoperfusion are less numerous. The indications are mostly poisoning with hepatotropic toxic substances, diseases of the liver and the abdominal organs, and prophylaxis of lymphogenic metastasis of tumour (during operations).

Purification of lymph by sorption is done by catheterization of the thoracic lymph duct on the neck and a vein. Lymph passes from the lymph duct into the column packed with the sorptive material and returns into the body through the vein.

Purification of lymph continues longer than that of blood (for at least 24 hours).

**Haemodialysis.** Removal of toxic substances from blood using a semipermeable membrane is called haemodialysis. The method

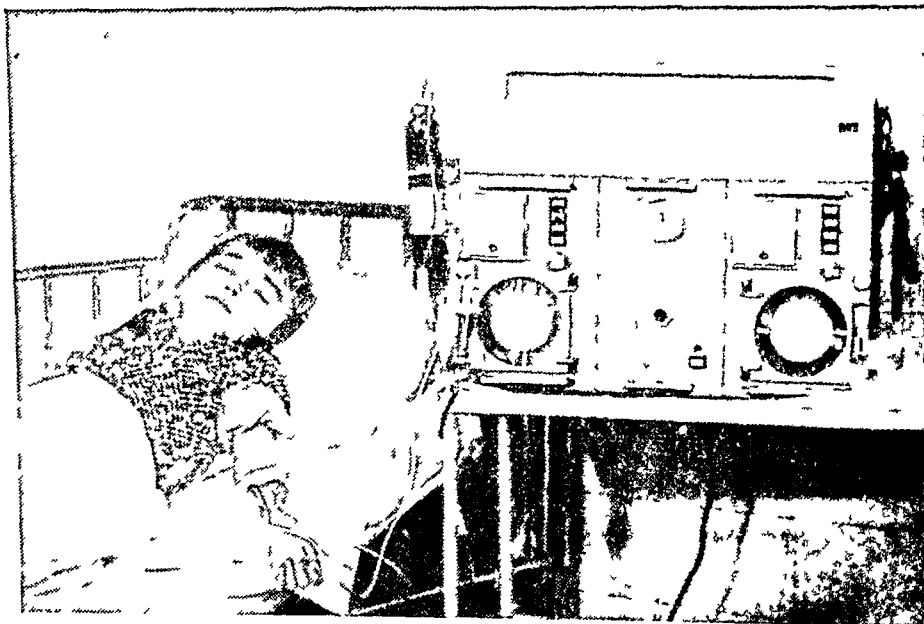


Fig 57 Haemoperfusion

is based on the difference (gradient) of concentrations of substances on either side of the membrane. As a rule, a toxic agent passes through the membrane in the direction of its smaller concentration. This principle is used in the apparatus known as an 'artificial kidney'. A semipermeable membrane is installed in a vessel, the dialysing liquid, simulating the blood with respect to its salt composition and osmotic properties and containing no toxic agent (or containing it in the concentration lower than in the blood), passes by one side of the membrane. The blood, from which the toxic agent is to be removed, moves by the other side of the membrane. Owing to this concentration gradient, the toxic substance passes the membrane in the direction of the dialysing solution, which carries it away. Another method to remove toxic agents from the blood is by increasing barometric pressure in that part of the dialyser where blood is contained. The pressure gradient forces water (and fine molecules) to pass through the membrane from the blood into the dialysing fluid, thus causing dehydration of the body.

The presence of toxic substances in the blood that can pass a dialysing membrane is the *indication* for haemodialysis. This condition occurs in renal and hepatorenal insufficiency and in hyperkalaemia of various genesis. Two main veins, or an artery and a vein, are used for carrying out dialysis. Blood from the vein (artery) is passed into a dialyser, and the dialysing solution is passed into the other chamber of the dialyser separated by a membrane. A counter-current prin-

ciple is used in the dialyser. Blood should be heparinized for haemodialysis.

**Peritoneal dialysis.** This is removal of toxic substances from the body through a natural semipermeable membrane, the peritoneum. The peritoneum is a barrier between the abdominal cavity and the inner medium of the body in the first instance between the lymph and blood. Transudation and absorption processes constantly occur in it. The peritoneum regulates the water-electrolyte composition of the blood, acid-base balance, protein metabolism, osmotic pressure of the blood, and eliminates various toxic substances, provided there is a gradient in their concentrations in the blood and in the abdominal cavity (see 'Haemodialysis').

Peritoneal dialysis is *indicated* for eliminating toxic substances, whose molecules are commensurable with the peritoneal pores, chronic and acute renal failure, hepatorenal insufficiency, exogenous poisoning, severe forms of diffuse peritonitis, and hyperhydration. The procedure is as follows. A catheter (catheters) is passed into the abdominal cavity (by puncturing it, or after a special operation). The dialysing solution, introduced into the abdominal cavity, absorbs toxic substances due to the difference in their concentrations. Whenever necessary, it is possible to withdraw liquid from the body by creating osmotic and oncotic pressure difference.

Two main types of peritoneal dialysis exist: permanent (pass-through) and fractional (intermittent). In the former case, the dialysing fluid is passed into the abdominal cavity through one catheter in a continuous stream and withdrawn from it through another catheter. One catheter is enough in the intermittent type of dialysis. The dialysing fluid is administered into the abdominal cavity and is allowed to remain there for a certain time (at least 40 minutes). The fluid is then withdrawn from the cavity and a fresh solution administered instead.

**Enterosorption.** This is the removal of toxic or infectious agents from the intestinal contents and from the internal medium of the body by sorbing substances taken per os. Like in haemoperfusion, carbon and resins are used for the purpose, but in this case they are used in the form of suspensions of powdered coal or water-soluble resins. The toxic agents are removed with the bowel contents, and also by their passage from the circulating blood into the bowel through the intestinal wall, which acts as a semipermeable membrane.

Exogenous peroral poisoning, diseases of the liver and renal failure are *indications* for enterosorption.

The technique is simple: the sorbing substance is taken per os or through a gastric tube.

**Replacement blood transfusion and plasmapheresis.** The detoxicating effect of this method is based on complete or partial replace-

ment of the patient's blood in cases with severe intoxication. Replacement transfusion of blood lessens intoxication, normalizes haemostasis, circulation and microcirculation, and improves the immunological status.

Progressive sepsis, septic shock and severe exogenous intoxications are *indications* for replacement transfusion of blood.

The procedure includes a puncture of the main vein, through which the child's blood is withdrawn and the donor blood infused. About 8-10 per cent of the circulating volume is withdrawn from the child during one session and the same amount is infused. From 50 to 150 per cent of the circulating volume can thus be replaced. The withdrawn blood can be centrifuged to separate the erythrocytes which can then be transfused back to the patient while the plasma should be taken from the donor (or albumin infused). This procedure is known as plasmapheresis.

## Chapter 31

### Intensive Therapy of Some Diseases of Newborns

#### HYALINE MEMBRANE DISEASE

Hyaline membrane disease (respiratory distress syndrome of the newborn) occurs in 0.5-1 per cent of neonates. The incidence varies with body weight: the smaller the weight, the higher the incidence.

*Aetiology and pathogenesis.* The leading factor in the onset of the disease is insufficiency of the surfactant system of the child. In most cases a sufficient amount of surfactant is present in the neonate lungs, which ensures effective respiration within several hours. But the synthesis of the surfactant, which is activated by methyltransferase, is probably discontinued after birth, while the phosphocholinesterase system of premature children is inadequate. The neonate therefore breathes freely only for a short period of time following his birth, but as the surfactant is gradually destroyed, the respiratory distress develops. The alveoli are distended during inspiration and collapse again during expiration. The work of breathing increases significantly because each breath consumes the same amount of energy as the first one. As the infant's energy resources are exhausted the collapse of the alveoli increases. The resistance of the pulmonary vessels increases, while the inflow of blood to the lungs decreases accordingly. The right-to-left shunting is again activated (first of all through Botallo's duct and the oval orifice). Increased pressure in the lesser circulation, hypoxia and acidosis promote the plasma passage through capillary walls and fibrin deposition (hyaline membranes) in the alveoli and the alveolar ductules.

*Clinical picture* The respiratory distress progresses in neonates with the hyaline membrane disease during the first hours following birth. The child becomes flaccid and assumes the batrachian (frog-like) position. General cyanosis develops. Tachypnoea increases gradually (to 60 and more breaths per minute), expiratory grunt is characteristic and is explained by the child's effort to expand the lungs by creating increased resistance to expiration. The yielding areas of the chest and the epigastrium are retracted during inspiration, which is a sign of decreasing lung compliance. Auscultation reveals weak respiration and diffuse crepitation at the end of expiration. Respiration is periodically arrested. In the absence of increased oxygen concentration in the inhaled air, the child develops cyanosis. The heart rate is usually high, but bradycardia (below 100 beats per minute) develops in cases with severe hypoxia. Disturbed peripheral microcirculation is manifested by pallor and fall of skin temperature.

Analysis of the gas composition of the arterial blood reveals hypoxaemia. In severe forms of the hyaline membrane disease  $\text{PaO}_2$  is usually below 45 mm Hg with air breathing. It increases insignificantly during breathing with mixtures containing increased oxygen concentration. Hypoxaemia is often attended by hypercapnia ( $\text{PaCO}_2$  is above 55 mm Hg) and acidosis (pH is below 7.25).

The electrolyte composition of plasma is usually normal, except that the potassium concentration increases in hypoxia-affected cells.

The test for foam resistance is specific. To 0.5 ml of isotonic sodium chloride solution added is 0.5 ml of gastric content and 1 ml of pure alcohol. The test tube is shaken energetically and the result is assessed in 15 minutes. The presence of surfactant causes the formation of stable bubbles on the surface of the solution. The test is negative in the absence of bubbling.

X-ray signs of the hyaline membrane disease are decreased transparency of the lungs (reticulogranular appearance) and the presence of light stripes in the lung root (with an air bronchogram).

*Treatment* The neonate with hyaline membrane disease should be placed in a couveuse at a temperature of 35°C (or higher). The air-oxygen mixture delivered into the couveuse should contain to 50-60 per cent of oxygen. If cyanosis persists and the partial tension of oxygen in the arterial blood does not rise above 50 mm Hg, spontaneous respiration with constant positive pressure in the airways should be conducted. The starting pressure should be 5 cm  $\text{H}_2\text{O}$ . The pressure should then be increased by 2-3 cm  $\text{H}_2\text{O}$  (with control of the gas composition of the blood) until  $\text{PaO}_2$  is stable at 60-70 mm Hg. The maximum pressure in the airways should not exceed 10-12 cm  $\text{H}_2\text{O}$ . As the condition of the neonate improves and  $\text{PaO}_2$  increases, the oxygen concentration in the breathing air should be



decreased to a non-toxic level (below 50 per cent), the pressure in the airways should then be lowered

If an attempted use of spontaneous respiration with constant positive airway pressure fails, and respiration is arrested or  $\text{PaCO}_2$  increases, the child's lungs should be ventilated artificially. The maximum values of ventilation for treatment of patients with severe hyaline membrane disease are as follows: pressure at the end of inspiration, 30-50 cm  $\text{H}_2\text{O}$ , positive pressure at the end of expiration from 8 to 10 cm  $\text{H}_2\text{O}$ , and the inspiration to expiration time ratio 3:1. It should be remembered that artificial ventilation at this rate can cause pathological changes and complications. These ratings should therefore be decreased as soon as possible.

The condition of the child with non-complicated hyaline membrane disease usually improves in 48-72 hours. During this time it is necessary to ensure parenteral administration of glucose, electrolytes and amino acid solutions to meet the child's demands for liquid and energy. Metabolic acidosis is corrected by intravenous administration of a 4 per cent sodium bicarbonate solution (2-4 mmole/kg body weight), the solution should be administered not faster than at a rate of 1 mmole per minute.

Many children with hyaline membrane disease develop hypovolaemia and hypotension. The deficit of circulating blood volume is replenished by transfusion of plasma or the formed elements (10 ml/kg). The haematocrit should be maintained at the level of 0.45-0.5 (45-50 per cent by volume). By the end of the second or third day of life the neonate should be trained for breathing without a ventilator so that he can breathe spontaneously with constant positive airway pressure. Further oxygen therapy should comply with the general requirements for this procedure. Infants, who had a severe form of hyaline membrane disease, can later grow normally.

### MECONIUM ASPIRATION SYNDROME

Aspiration by the foetus of the amniotic fluid containing admixtures of meconium is the result of intranatal hypoxia and increased respiratory activity of the foetus. Hypoxia can develop gradually due to placental insufficiency, or it may occur acutely as a result of umbilical prolapse or placental detachment. Respiratory distress can develop immediately after birth or gradually, as meconium particles pass to the peripheral parts of the airways. Infants develop tachypnoea with involvement of the accessory muscles. Many rales of various caliber can be auscultated in the lungs. The x-ray picture is characterized by infiltration of the lung roots, fan-like patterns of atelectasis, and emphysematous sites (caused by air retention). The prevailing factor is in most cases overdistension of the lungs in the presence of vast atelectases. The diaphragm contours on x-ray

pictures are therefore level, the intercostal spaces are protruded. The study of the acid-base balance and the gas composition of blood reveals hypoxaemia and mixed acidosis.

The efficacy of treatment depends on as early as possible removal of the aspirated material from the airways. The studies have shown that timely removal by suction of the tracheal contents decreases lethality in this group of patients 2 or 3 times. The resuscitation measures should therefore be started with laryngo- or bronchoscopy, and irrigation of the trachea and bronchi with an isotonic sodium chloride solution or sodium bicarbonate solution with subsequent aspiration of excess liquid. The gastric contents stained with meconium should also be removed. Intensive therapy includes control of gas composition of the blood, correction of acidosis, inhalation of moist oxygen-air mixtures, and preventive use of antibiotics.

In severe cases of meconium aspiration, the child is given artificial ventilation of the lungs. The difficult adaptation of child with the meconium aspiration syndrome to a ventilator usually requires the use of muscle relaxants. The maximum pressure during artificial lung ventilation in such patients is 30-35 cm H<sub>2</sub>O and the respiration rate, 40-45 breaths per minute, which facilitates removal of carbon dioxide mainly through the non-involved portions of the lungs. To prevent air retention in the lungs and emphysema-tous changes, the inspiration to expiration ratio should not be higher than 1 : 2. The child with the meconium aspiration syndrome should be given artificial lung ventilation during 2 to 5 days.

### APNOEA OF NEWBORNS (APNOEA NEONATORUM)

Periodical attacks of apnoea (cessation of spontaneous respiration for 10-30 seconds) usually occur in neonates with small body weight and are usually associated with functional immaturity of the central nervous system. Among other causes of apnoea, metabolic acidosis, exposure to cold, hypoglycaemia, hypocalcaemia, hyperbilirubinaemia, and anaemia deserve mentioning. Occasional attacks of apnoea can be the result of previous asphyxia or small haemorrhages into the brain. Cessation of respiration for more than 10 seconds is usually attended by bradycardia, apnoea lasting for more than 20 seconds causes severe hypoxaemia. Children showing the tendency to apnoea should be monitored (their respiration and heart rates should be controlled) to ensure timely diagnosis and prevention of severe complications.

A common resuscitation measure during apnoea of neonates is massage of the chest. If it fails, artificial ventilation of the lungs should be ensured using a face mask of the anaesthesia apparatus or a breathing bag. Artificial ventilation in children with frequently recurring attacks of apnoea (several attacks an hour), or in whom

these attacks are accompanied with gas exchange and circulatory disorders, should be controlled automatically. If the condition of the child is not aggravated with concurrent diseases of the respiratory system, the rate of artificial ventilation should be 25-30 per minute, while the maximum inspiration pressure should be 15-18 cm  $H_2O$ , the inspiration to expiration ratio being 1:2. End expiratory pressure should be normal. After the condition of the child has stabilized, intermittent mandatory ventilation should be applied with the respiration rate decreased to 1-2 per minute. Assisted respiration with constant positive airway pressure of about 3-5 cm  $H_2O$  is effective in some cases.

### PNEUMOTHORAX AND PNEUMOMEDIASTINUM

Spontaneous pneumothorax and pneumomediastinum often occur during the first breaths of a neonate with rigid lungs. Excessively active respiratory movements overdistend normally ventilated alveoli and cause their rhexis. This usually occurs in infants with hyaline membrane disease, aspiration syndrome, pneumonia, and hyperplasia of the lungs. A small volume of free air in the chest can be found in 1-2 per cent of practically healthy neonates. The incidence of these complications has considerably increased now due to incorporation of new methods for intensive respiratory therapy based on mandatory inflation of the lungs. Pneumothorax occurs in 5-40 per cent of children during artificial lung ventilation with positive end expiratory pressure.

Pneumomediastinum and pneumothorax have a common pathogenesis. As alveoli break, the air penetrates the connective tissue and passes along the blood vessels toward the lung root, from where it enters the mediastinum and the pleural cavity. In case of rhexis of the alveoli, located immediately beneath the pleura, the air can pass directly into the pleura. The presence of a small amount of air in the mediastinum or the pleural cavity can be symptomless and there may be no obvious indications for intensive therapy. But the accumulation of air in the pleural cavity, especially in the presence of positive pressure (valvular pneumothorax), rapidly worsens the child's condition to endanger his life. Tachypnoea increases suddenly and generalized cyanosis develops. The involved side of the chest may be protruded. The mediastinal organs are displaced to the opposite side, which can be revealed by displacement of the area of cardiac dullness and the apex beat. Percussion reveals the presence of band-box sound over the involved side of the chest, auscultation reveals the absence or strongly diminished respiration in the involved side. The heart sounds are dull and the heart rate is fast.

X-ray examinations reveal the presence of free air outside the partly collapsed lungs, the absence of the bronchial pattern outside

the lungs, and displacement of the mediastinal organs and the diaphragmatic cupola.

Development of intense pneumothorax requires immediate thoracocentesis to withdraw the air from the pleural cavity. The 4th intercostal space is commonly punctured in neonates (in the anterior axillary line). The continuing entrance of air into the pleural cavity is an indication for draining the cavity and connecting the catheter to a source of vacuum (constant rarefaction of 10-20 cm H<sub>2</sub>O).

As distinct from pneumothorax, pneumomediastinum almost never causes severe disorders in the vital functions. Chest x-ray reveals the presence of an air envelope around the heart. The air is often seen between the silhouette of the heart and the thymus, which is slightly raised.

Pneumomediastinum is treated by periodical inhalation of 100 per cent oxygen.

### SHOCK

Shock is a severe circulatory disorder, during which the cardiac output cannot meet the demands of the body. Hypovolaemic shock can be caused by acute loss of blood in umbilical rupture, placental detachment, retraction of the internal organs, and similar conditions. Shock often occurs in asphyxia, hyaline membrane disease, in severe intoxications, and in the presence of excessively high pressure in the end of expiration during artificial lung ventilation.

The clinical manifestations of shock in neonates are tachycardia (160 beats per minute), tachypnoea, pallor, and subnormal temperature of the skin. Diuresis is either absent or decreased considerably. The arterial pressure is another important diagnostic sign. The lower limit of systolic pressure in a newborn weighing over 2500 g is 60 mm Hg.

A child in the state of shock should be placed in a couveuse or exposed to radiant heat so that his body temperature should be maintained at 36°C. The heart rate, the skin temperature, and (if possible) arterial and central venous pressure should be monitored. Hourly diuresis should be controlled.

To prevent hypoxaemia air-oxygen mixtures should be given to inhale, the lungs should be ventilated artificially whenever necessary. The volume of circulating blood should be re-established by intravenous injections of blood or plasma (10 ml/kg, within 2-5 minutes) and Ringer lactate (5-10 ml/kg). The infusion of liquids should be controlled by the arterial and central venous pressure. Metabolic acidosis should be corrected by administering sodium hydrocarbonate solution. In order to improve the contraction of the myocardium and to increase the cardiac output, dopamine should be administered at a rate of 6-8 µg/kg per minute.

## Chapter 32

## Intensive Post-operative Therapy

Operative trauma and anaesthesia cause considerable changes and disorders in the body, and some authors consider this complex of changes as a separate 'post-operative disease'. Disturbances in the central nervous system, respiratory organs, cardiovascular system, gastro-intestinal tract, and in the urinary system, and also serious biochemical shifts in practically all types of metabolism, are observed during this period. Correct management of the post-operative patient requires special attention on the part of the anaesthesiologist, which is later rewarded by a considerable improvement of the child's condition and his recovery.

NON-COMPLICATED POST-OPERATIVE  
(POST-ANAESTHESIA) PERIOD

The main objects of management of a non-complicated post-operative period are prevention and treatment of pain syndrome, therapy of respiratory insufficiency, normalization of the acid-base balance, and replenishment of energy loss of the body.

Any operative intervention involves a more or less pronounced *pain syndrome*. Pain is not only the source of physical sufferings of a child, it causes some post-operative respiratory complications. Pain decreases the respiratory excursions of the chest and the diaphragm and interferes with evacuation of secretion from the tracheo-bronchial tree (difficult expectoration), which can after all cause atelectasis and pneumonia. The pain syndrome is most pronounced after operations on the thoracic and abdominal organs, and also in vast orthopaedic operations.

Pain is controlled mainly by analgesics, usually by promedol. Compared with the other drugs of the morphine group it inhibits respiration less significantly and can be given to children of any age. If pain is especially severe, promedol should be administered together with antihistaminics (diprazin, suprastin, dimedrol, etc.), or with small doses of droperidol, which intensifies and prolongs the narcotic effect. It should be remembered that habituation to promedol and other narcotic drugs soon develops, and they should therefore be used with great care only during the first 24-48 hours post-operative. Their use should be suspended when pain lessens. If pain is not severe, it can be eliminated by intramuscular administration of analgin, which should also be better used with antihistaminics. This preparation is often very effective with newborns and nurslings.

If the operative injury is significant (in operations on thoracic or abdominal organs) pain should be controlled by catheterization of the epidural space for fractional administration of anaesthetics (trimecaine, lidocaine, dicaine) for 2 or 3 days. Epidural anaesthesia during post-operative period promotes normalization of the external respiratory function and rapid re-establishment of intestinal peristalsis.

A good sedative and analgesic effect during post-operative period can be attained with acupuncture (electroacupuncture). Acupuncture can be used instead of analgesics in about 60-70 per cent of cases (or at least the doses of analgesics can be significantly decreased).

*Respiratory insufficiency* during post-operative period is prevented and treated by maintaining free patency of the tracheobronchial tree, by improving bronchial drainage and also by blood oxygenation. Inhalation therapy is obligatory for all children after endotracheal anaesthesia in order to prevent subglottic laryngitis, because the endotracheal tube can cause inflammation of the loose submucous cellular tissue of the larynx. A warm isotonic sodium chloride solution, a 4 per cent sodium bicarbonate solution, or mineral water (Borzhomi) should be used for inhalations, 2-3 times a day. In the presence of inflammation of the upper airways good effect is attained by steam-oxygen inhalations with medicinal herbs (sage, thyme, plantain, coltsfoot, wild rosemary, etc.), which are alternated with aerosols of proteolytic enzymes (trypsin, chymotrypsin, mucosolvin, etc.). It should however be remembered that prolonged use of enzymes thins sputum facilitating its expectoration but may impair the drainage of the bronchi due to affection of the epithelial cilia, allergic reactions are also possible. Inhalations should therefore be conducted not more than 2 or 3 times a day, for 2-3 days. Sputum is also thinned if the patient drinks much water (mineral water Borzhomi, Djermuk, Essentuki, etc.). If the patient cannot drink, e.g. after operation on the gastrointestinal tract, in intestinal paresis, etc., adequate amount of liquid should be given parenterally.

If mucus is retained in the upper airways, it should be removed by suction using a thin sterile catheter (better with direct laryngoscopy). The catheter should remain in use of a particular patient till the end of treatment and kept in an antiseptic solution. In hyptil the end of treatment and kept in an antiseptic solution. In hypersecretion mucus should be removed by suction at 30-40 minute intervals, and obligatory after each inhalation.

Oxygen therapy is obligatory for all children during the first 2-3 days after operation on the thoracic and abdominal organs, other children should be given oxygen therapy for special indications, i.e. in the presence of first signs of respiratory insufficiency. Among other methods for oxygen therapy of non-complicated post-operative periods a very simple method should be used: inhalation of humidified oxygen through nasal catheters. Elastic tubes, richly lubricated

with glycerol, are passed through the nose into the nasopharynx. As pure oxygen is passed through the catheters, its concentration in the inhaled mixture is 35-54 per cent. This concentration is optimum for substantial oxygenation, it also prevents the danger of toxic effect of oxygen given at concentrations of 60-70 per cent for a long time. Only humidified and warmed (to the temperature of the body) oxygen can be used for oxygen therapy. If a humidifier or pre-heater are not available, a Bobrov bottle can be used. Oxygen is passed through hot water and is thus warmed to 34-36°C.

Remedial exercises, respiratory exercises, and the active behaviour of a child are very important for prevention of respiratory disorders.

*Homeostasis* is significantly impaired in children after vast operative interventions. This should be corrected by appropriate intensive therapy because otherwise disordered homeostasis can cause paresis of the stomach and the intestine, upset the hepatic function, and decrease the diuresis, thus interfering with removal of metabolites from the body. The condition of the operated child can be endangered if these pathological changes are not corrected in due time.

Correct management of the post-operative period is impossible without timely and thorough correction of the acid-base equilibrium and the water-electrolyte balance.

Development of metabolic acidosis during the post-operative period is the protective response of the body to operative injury. But significant changes in the acid-base balance cause severe disorders in the intracellular metabolism, inactivation of most key enzymes, and hence, functional disorders in the vital organs and systems. Metabolic acidosis is corrected with control of the acid-base balance by intravenous administration of a 4 per cent sodium bicarbonate solution. But if the acid-base balance cannot (for some reason or another) be controlled, tentative calculations can be done using the results of numerous clinical observations. 3 ml of a 4 per cent sodium bicarbonate per kg body weight are required after operations lasting to 90 minutes, if the operations last for longer time, the dose should be increased to 4 ml. If post-operative acidosis is due to disordered external respiratory function (increased  $PCO_2$  of blood) rather than metabolic disorders (BE changes insignificantly), sodium bicarbonate solution is not very effective and its administration is not recommended. Measures should be taken to eliminate respiratory insufficiency. These include adequate oxygenation, spontaneous respiration with constant positive airway pressure, and artificial lung ventilation in severe cases.

Sodium bicarbonate alone can normalize the acid-base balance in rare cases. Acidosis develops again in 30-40 minutes unless its causes are eliminated. The therapy of the disordered acid-base balance should therefore be complex and include adequate analgesia,

adequate oxygenation, timely replenishment of blood loss and the blood circulating volume, and correction of the ionic balance and the renal function

Acidosis usually occurs only during the first post-operative day, on the next day it is followed by metabolic alkalosis, which is mainly connected with the loss of potassium and the chloride ion (hypokalaemic and hypochloraemic alkalosis). Metabolic alkalosis can be corrected by intravenous administration of potassium chloride solution. The needed quantity depends on the potassium deficit and the physiological loss of potassium—2 mmole/kg a day (see Chapter 25). For example, if the potassium content of the blood serum of a child is 3 mmole/l, the deficit is 2 mmole/l (the normal average is 5 mmole/l). The total deficit and the daily requirements are calculated with reference to the extracellular fluid, which makes 0.4 of body weight. Thus, the amount of potassium that should be administered to a child can be calculated from the formula:  $\text{body weight (kg)} \times (0.4 \times P_d + 2P_p)$ , where  $P_d$  is potassium deficit and  $P_p$  is potassium physiological loss. For example, the potassium deficit of a child weighing 10 kg is 2 mmole/l, then the amount of potassium that should be administered parenterally is  $10 \times (0.4 \times 2 + 2) = 28$  mmole. To that end, 28 ml of a 7.5 per cent potassium chloride solution, whose 1 ml contains 1 mmole of potassium, should be diluted 1:10 in a 5 or 10 per cent glucose solution containing insulin (1 unit per 3 g of glucose). This solution should be administered by drip infusion at a rate not exceeding  $\frac{1}{5}$  of the daily dose per hour. Rapid administration of the solution can cause a cardiac arrest.

Correction of potassium metabolic disorders during the post-operative period is obligatory because hypokalaemia causes persistent intestinal paresis, disorders in the cardiac function and conduction in the neuromuscular synapses, and some other serious disorders.

If it is impossible to determine the electrolyte level in the blood, potassium chloride should be administered in the dose equal to the daily demand for potassium of infants (about 3 mmole/l).

*Hyponatraemia* can occur during the post-operative period, it should also be corrected. Sodium chloride solution of various concentrations (see Chapter 25) should be used for the purpose. The solution may contain 154, 513, or 885 mmole/l of sodium chloride (0.9, 3 and 5 per cent solutions, respectively). The amount of solutions to be administered (ml) are calculated from the following formula

$$\frac{\text{total sodium deficit} \times 1000 \text{ ml}}{\text{sodium content (mmole/l) of a given solution}}$$

The total sodium deficit is equal to the deficit of sodium multiplied by the volume of extracellular fluid (see above). The sodium



deficit is the difference between its normal content (140 mmole/l) and the actual content in a given patient. The calculated dose should be administered slowly, at a rate of 12-15 drops per minute. The tendency to hyponatraemia is especially vivid in newborns, all pathological losses in them (especially the loss of the gastric contents) should therefore be replenished by Ringer's solution (in equivalent amounts).

The operative injury and fasting during the early post-operative period cause considerable changes in the protein metabolism, which are manifested by *hypo- and dysproteinaemia*, increasing concentration of test nitrogen, nitrogen of amino acids and urea in the blood. Excretion of total nitrogen and nitrogen of urea increases with the urine, and the nitrogen balance becomes negative. This is the result of increased catabolism, which is characteristic of most stress conditions, the post-operative period included.

The considerable loss of protein can in most cases be replenished during the post-operative treatment by parenteral administration of protein hydrolysates or equilibrated amino acid mixtures in amounts sufficient to meet the protein demands of the body and to equilibrate the upset balance between separate amino acids in the liver and other organs. Aminosol, aminon, alvesin, moriamin and vamin are used for correction of the protein metabolism in operated children. These preparations contain all main amino acids in the required proportions and can maintain the protein metabolism at normal level.

The dose of a protein preparation depends on the degree of metabolic disorder. If the disorder is compensated, 1 g/kg of protein a day should be administered. Decompensation of protein metabolism, which is manifested by hypoproteinaemia, decreased albumin-globulin ratio, and increased urea content in the daily urine, should be treated by increased protein doses (3-4 g/kg a day), and anticatabolic therapy (retabolil, nerobolil, 25 mg intramuscularly, once during 3-5 days).

Another important factor of adequate parenteral nutrition is the supply of sufficient *energy material* in the form of carbohydrates and fats, because the energy demand during post-operative period increases considerably. The energy demands should be met adequately, because not only the proteins taken with food but also proteins of own tissues will be consumed for conversion into energy in the absence of the main source of energy (fats and carbohydrates). The utilization of own tissue proteins intensifies hypoproteinaemia of the post-operative period. The exogenous energy supply in the form of fats and carbohydrates thus decreases the decomposition of proteins and produces the nitrogen-saving effect. The mixture of amino acids is assimilated to the optimum degree if the ratio of the calories to the amount of the taken nitrogen (in grams) is 150:1.

Fats and carbohydrates are the main suppliers of ATP of the cell, its main source of energy. In the absence of fats and carbohydrates the cell consumes intensively amino acids. The functional condition of the protein-synthesizing apparatus also depends on a continuous supply of energy-giving material to the cell. About 4-5 g of endogenous nitrogen can be saved during medium-gravity operations by parenteral administration of glucose. This amount of nitrogen is equivalent to 25-31 g of protein.

During early post-operative period when normal nutrition (eating) is either restricted or completely impossible, the operated child should be given parenteral nutrition by infusion therapy. The necessary amounts of amino acids, caloric demands, quantities of electrolytes and vitamins, and also the total amount of liquid, should be calculated very accurately, especially for newborns and infants.

The daily caloric demand of infants ageing under 1 is 110-130 kcal/kg, of infants between 1 and 3, 80-90 kcal/kg, between 4 and 5, 70-80 kcal/kg, between 10 and 12, 50-60 kcal/kg, and of older children 35-45 kcal/kg.

Energy demands during parenteral nutrition cannot be met without correction of fat metabolism. In order to attain this and also to increase the caloric intake with parenteral nutrition, fat emulsions containing polyunsaturated fatty acids are now used. The emulsions are prepared from cotton-seed and soya bean oil (lipifizan, lipofundin, intralipid). Fat is a high-caloric material, but its utilization is only possible with correct dosage and rate of administration (1.5-3 g/kg a day, 0.2 g/kg per hour). Moreover, fat emulsions should not be administered with concentrated glucose solutions, or immediately before or after glucose administration.

Contraindications for administration of fat emulsions are hepatic insufficiency, lipaemia, hypoxaemia, shock, thrombohaemorrhagic syndrome, microcirculatory disorders, brain oedema, and haemorrhagic diathesis.

When administering parenteral nutrition, it is necessary to administer also optimum doses of vitamins, which are involved in many metabolic processes since they act as co-enzymes in the reactions of energy utilization. The amounts of vitamins that should be administered per each 100 kcal are as follows: ascorbic acid 3-5 mg, co-carboxylase 20-30 mg, thiamine 0.1 mg, riboflavin 0.1 mg, pantothenic acid 0.3 mg, choline 5-10 mg, nicotinic acid 0.7 mg, pyridoxine 0.1 mg, folic acid 2 mg, and cobalamine 0.04 mg.

The ingredients of the parenteral nutrition should be balanced. The optimum ratio of proteins to fats and to carbohydrates should be 1:1.8:6:6. In order to split proteins, fats and carbohydrates, and to involve them in various syntheses, a certain amount of water is necessary, namely, 1 ml of water per each kilocalorie.

Thus, when planning infusion therapy during post-operative

period, a daily programme should be calculated, in which all requirements for electrolytes, proteins, fats, carbohydrates, vitamins, and water should be thoroughly balanced. Total parenteral nutrition should be given only during the first post-operative days. When the child is able to eat, the quantity of ingredients given parenterally should be decreased accordingly, i.e. total parenteral nutrition should be replaced by partial parenteral nutrition, which is a supplement to the food taken by the child per os.

### COMPLICATED POST-OPERATIVE (POST-ANAESTHESIA) PERIOD

Early post-operative period can be complicated by changes in the central nervous system, by respiratory and circulatory disorders and disturbances in the gastrointestinal function. The common complication on the part of the central nervous system is *late recovery from anaesthesia*. It can be due to overdosage of narcotic substances, hypercapnia caused by inhibition of respiration by large doses of promedol, phentanyl and muscle relaxants, and also inadequate artificial ventilation of the lungs and pronounced metabolic acidosis due to excess accumulation of lactic, pyruvic and other acids in the child's body.

Overdosage of narcotic substances is diagnosed by deep narcotic sleep (absence of the pupillary and corneal reflexes), EEG findings, and also by the quantity of preparations used for narcosis. Antidotes to promedol and phentanyl should be used in such cases (nalorphine, naloxon). Adequate oxygenation (assisted spontaneous or artificial ventilation) should be ensured.

Metabolic acidosis should be corrected by intravenous administration of sodium bicarbonate solution. It is necessary simultaneously to improve the function of the urinary system. The urinary bladder should be catheterized, the circulating volume of blood replenished, and aminophylline and, if necessary, furosemide administered intravenously.

Prolonged unconsciousness, increased muscular tone, convulsions and pathological reflexes indicate hypoxic affection of the brain (post-hypoxic encephalopathy). This is caused by oxygen deficit due to a pronounced and prolonged fall of arterial pressure, inhibited respiration, or obstruction of the airways.

Treatment: oxygen therapy, hyperbaric oxygenation, artificial lung ventilation (if necessary), control of brain oedema, using protease inhibitors (contrikal, gordox), diuretics (lasix, mannitol, urea), glucocorticoids, and craniocerebral hypothermia.

The main complications of the respiratory function and the cardiovascular system, their diagnosis, prevention, and treatment were described in Chapter 12.

Upset gastrointestinal function during early post-operative period is manifested mainly by vomiting and paresis of the intestine and the stomach. A permanent thin tube should in such cases be introduced into the stomach to withdraw congestive contents. Measures should be taken to re-establish peristalsis by correcting electrolyte disorders and acid-base balance, administering proserine, parane-phric novocaine block, and prolonged epidural anaesthesia.

## Appendix

*1 Main Drugs Used for Anaesthesia and Intensive Therapy of Infants and Children*

| Preparation                                       | Form        | Dose   |                  |             |
|---|-------------|--------|------------------|-------------|
|   |             |        | under<br>1 month | 6 months    |
| <i>I Narcotics</i>                                |             |        |                  |             |
| Hexenal, 1%                                       | solution    | single |                  |             |
| Thiopental sodium, 1%                             | solution    | single |                  |             |
| Sodium oxybate, 20%                               | solution    | single | 50-100 mg/kg     |             |
| Sombrevin (epontol)                               | solution    | single |                  |             |
| Ketamine, 5%                                      | solution    | single |                  |             |
| <i>II Hypnotics</i>                               |             |        |                  |             |
| Phenobarbital                                     | tablets     | single | 0.005            | 0.01        |
| Chloral hydrate                                   | powder      | single | 0.1              | 0.15        |
| Barbital  | powder      | single | 0.01             | 0.04        |
| Medinal   | tablets     | single | 0.01             | 0.03        |
| <i>III Psychotropic drugs</i>                     |             |        |                  |             |
| Aminazine, 2.5%                                   | solution    | single |                  | 0.1 ml      |
| Droperidol, 0.25%                                 | solution    | single |                  | 0.3-        |
| Diazepam, 0.5%                                    | solution    | single |                  | 0.1-0.2 ml  |
| Propazine, 2.5%                                   | solution    | single |                  | 0.1 ml      |
| Fluphenazine, 0.25%                               | solution    | single |                  | 0.1 ml      |
| Benactyzine (amizyl)                              | tablets     | single |                  |             |
|   | powder      |        |                  |             |
| <i>IV Narcotic analgesics</i>                     |             |        |                  |             |
| Dextiomoramide<br>(palfium)                       | solution    | single |                  | 0.1-0.2 ml  |
| Promedol 1%                                       | solution    | single |                  |             |
| Omnopon, 1%                                       | solution    | single |                  |             |
| Phentanyl   | solution    | single |                  |             |
| <i>V Non-narcotic analgesics and antipyretics</i> |             |        |                  |             |
| Amidopyrine, 4%                                   | solution    | single | 0.3-0.5 ml       | 0.5-1.0 ml  |
| Amidopyrine                                       | suppository | single | 0.10 g           | 0.15 g      |
| Analgin, 50%                                      | solution    | single | 0.03-0.05 ml     | 0.05-0.1 ml |
| Acetylsalicylic acid                              | tablet      | single | 0.05 g           | 0.05-0.1 g  |

| Age                    |                      |                        | Administration                         | Notes                                  |
|------------------------|----------------------|------------------------|--|--|
| 1-3 years              | 4-7 years            | above 7 years          |  |  |
|                        | 8-10 mg/kg           |                        | intravenous                            | dilute with distilled water before use |
|                        | 5-8 mg/kg            |                        | intravenous                            | dilute as hevenal                      |
|                        | 100-150 mg/kg        |                        | intravenous<br>intramuscular<br>per os |  |
|                        | 5-15 mg/kg           |                        | intravenous                            | used for minor                         |
|                        | 6-12 mg/kg           |                        | intramuscular                          | (out-patient) opera-                   |
|                        | 2-3 mg/kg            |                        | intravenous                            | tions                                  |
| 0 02-0 03              | 0 03-0 04            | 0 04-0 05              | per os                                 | to prevent convul-                     |
| 0 2-0 3                | 0 3-0 4              | 0 4-0 5                | by enema                               | sions                                  |
|                        |                      |                        |  | dilute with distilled                  |
|                        |                      |                        |  | water to make                          |
|                        |                      |                        |  | 2 and 3% solutions                     |
| 0 07-0 1               | 0 15-0 25            | 0 3                    | by enema                               |  |
| 0 07-0 1               | 0 15-0 25            | 0 3                    | by enema                               |  |
| per year of age        |                      |                        | intravenous                            | used per se and in                     |
| 0 5 mg/kg              |                      |                        | intramuscular                          | lytic mixture                          |
|                        |                      |                        | intravenous                            | can be administe-                      |
| per year of age        |                      |                        |  | red again in 2-3 hrs                   |
|                        |                      |                        | intravenous                            | for convulsive syn-                    |
| per year of age        |                      |                        | intramuscular                          | drome and anxiety                      |
|                        |                      |                        | intramuscular                          | less toxic than ami-                   |
|                        |                      |                        |  | nazine                                 |
| per year of age        |                      |                        | intramuscular                          | antiemetic                             |
| $\frac{1}{3}$ tablet   | $\frac{1}{2}$ tablet | 1 tablet               | per os                                 | can be used as                         |
|                        |                      |                        |  | sedative                               |
| 0 2-0 4 ml             | 0 5-0 8 ml           | 0 9-1 ml               | subcutaneous                           | twice a day maxi-                      |
|                        |                      |                        |  | mum                                    |
| 0 1 ml per year of age |                      |                        | intravenous                            | respiration can be                     |
|                        |                      |                        | intramuscular                          | depressed                              |
|                        |                      |                        | subcutaneous                           |  |
| 0.1 ml per year of age |                      |                        | same                                   | same                                   |
| 0 008-0 01 mg/kg       |                      |                        | same                                   |  |
| 2 0-4 0 ml             | 4 0-6 0 ml           | 1.0 ml per year of age | intravenous                            | for pyrexia can be                     |
|                        |                      |                        | intramuscular                          | given in 1-2 hrs                       |
| 0 15-0 2 g             | 0 25-0 3 g           | 0.3-0 5 g              | per rectum                             | same                                   |
|                        |                      |                        | intramuscular                          | same                                   |
| 0 1 ml per year of age |                      |                        | intravenous                            |  |
|                        |                      |                        | per os                                 | for hyperpyrexia                       |
| 0 1-0 25 g             | 0 25-0 5 g           | 0 5 g                  |  |  |

| Preparation  | Form     | Dose   |                  |               |
|--|----------|--------|------------------|---------------|
|  |          |        | under<br>1 month | 6 months      |
| <i>VI Cholinolytics</i>  |          |        |                  |               |
| Atropine, 0.1%   | solution | single | 0.05 ml          |               |
| Methacine, 0.1%  | solution | single | 0.02-0.03 ml     | 0.05-0.1 ml   |
| Platyphylline, 0.2%  | solution | single | 0.1 ml           | 0.2 ml        |
| Scopolamine, 0.05%   | solution | single | 0.1 ml           | 0.1 ml        |
| <i>VII Ganglioblockers</i>                                     |          |        |                  |               |
| Arfonad, 1%  | solution | single |                  |               |
| Benzohexonium, 2.5%  | solution | single | 0.05-0.1 ml      |               |
| Pentamine, 5%  | solution | single | 0.5 mg/kg        | 0.5-1.0 mg/kg |
| <i>VIII Spasmolytics</i>                                       |          |        |                  |               |
| Aminophylline, 24%   | solution | single |                  |               |
| Aminophylline, 2.4%  | solution | single | 0.2-0.3 ml       | 0.5-1.0 ml    |
|  |          |        |                  |               |
| Nospa  | solution | single | 0.3-0.5 ml       | 0.5-0.8 ml    |
| Papaverine, 2%   | solution | daily  | 0.2 ml           | 0.4 ml        |
| <i>IX Antihypertensive preparations and alpha-adrenolytics</i> |          |        |                  |               |
| Magnesium sulphate, 25%  | solution | single | 1.0              |               |
| Reserpine, 0.1%  | solution | single | 0.03-0.05 ml     | 0.05-0.08 ml  |
| Dibazol, 0.5%  | solution | single | 0.1 ml           | 0.2 ml        |
| Tropaphen, 1%  | solution | single | 0.1 ml           | 0.2 ml        |
|  |          |        |                  |               |
| <i>X Vasopressors (alpha-mimetics)</i>                         |          |        |                  |               |
| Epinephrine, 0.1%  | solution | single | 0.1 ml           | 0.1-0.15 ml   |
| Notepinephrine, 0.2%   | solution | single |                  |               |
| Mesaton (phenylephrine hydrochloride), 1%                      | solution | single |                  |               |
| Ephedrine, 5%  | solution | single |                  |               |

*Continued*

| Age                     |            |                  | Administration                               | Notes  |
|-------------------------|------------|------------------|--|--|
| 1-3 years               | 4-7 years  | above<br>7 years |  |  |
| 0 1 ml per year of age  |            |                  | intravenous<br>intramuscular<br>subcutaneous | for severe bradycardia the dose can be increased 2-3 times   |
| 0 1 ml per year of age  |            |                  | subcutaneous<br>intravenous<br>intramuscular |  |
| 0 25-1 0 ml             | 1 0-1 2 ml | 1 2-1 5 ml       | subcutaneous                                 |  |
| 0 1-0 2 ml              | 0 2-0 3 ml | 0 3-0 5 ml       | subcutaneous                                 |  |
| 0 1-0 2 ml/kg           |            |                  | intravenous<br>intramuscular                 | can be given repeatedly in 1-2 hrs   |
| 0 2-0 3 ml              | 0 3-0 5 ml | 0 5-1 0 ml       | intravenous<br>intramuscular                 |  |
| 1 5-2.5 mg/kg           |            |                  | intravenous<br>intramuscular                 |  |
|                         |            |                  |  |  |
| 0 1 ml per year of age  |            |                  | intramuscular                                | intravenous drip with glucose should be preferred, slow infusion with control of arterial pressure |
| 1 0 ml per year of age  |            |                  | intravenous                                  |  |
| 0 8-1 0 ml              | 1 0-1 5 ml | 1 5-2 0 ml       | intravenous, slow<br>subcutaneous            |  |
| 0 5-0 6 ml              | 0 6-0 8 ml | to 1 ml          |  |  |
| ml per year of age      |            |                  | intramuscular                                | for disordered peripheral circulation  |
| 0 1-0 2 ml              | 0 2-0 3 ml | 0 3-0 4 ml       | intramuscular                                |  |
| 0 4 ml                  | 0 5-0 8 ml | 0 8-1 0 ml       | intramuscular                                |  |
| 0 2-0 4 ml              | 0 5-0 8 ml | 0 8-1 0 ml       | intramuscular                                |  |
| 0 2-0 3 ml              | 0 3-0 5 ml | 0 5-0 8 ml       | subcutaneous<br>intravenous                  | the dose can be increased 2-3 times for cardiac arrest can be given repeatedly at 3-4 hr intervals |
| 0 05 ml per year of age |            |                  | same   |  |
| 0 1 ml per year of age  |            |                  | same   |  |
| 0 1 ml per year of age  |            |                  | same   |  |



| Preparation                                 | Form                | Dose   |                          |                   |
|---|---------------------|--------|--------------------------|-------------------|
|   |                     |        | under<br>1 month         | 6 months          |
| <i>XI Antiarrhythmics and beta-blockers</i> |                     |        |                          |                   |
| Dilantin (phenytoin), tablet<br>1 mg        |                     | single | $\frac{1}{5}$ tbl        | $\frac{1}{5}$ tbl |
| Inderal (propanolol hydrochloride), 5 mg    | ampoule             | single | 0 2-0 5 ml               | 0 2-0 5 ml        |
| Novocainamide, 10%                          | solution            | single |                          |                   |
| <i>XII Beta-stimulants</i>                  |                     |        |                          |                   |
| Alupent                                     | solution            | single | 0 2-0 3 ml               | 0 2-0 3 ml        |
| Novodrin                                    | tablet              | single | $\frac{1}{4}$ tbl        | $\frac{1}{4}$ tbl |
| <i>XIII Cardiac glycosides</i>              |                     |        |                          |                   |
| Digoxin                                     | tablet,<br>solution | daily  |                          | 0 05-0 075        |
| Corglycon, 0 06%                            | solution            | single | 1-2 drops                | 0 05 ml           |
| Strophanthin, 0 05%                         | solution            | single | 1 drop                   | 0 05 ml           |
| <i>XIV Diuretics</i>                        |                     |        |                          |                   |
| Aldactone (spironolactone)                  | tablet              | single |                          | 0 01 g            |
| Glycerol                                    |                     | single | $\frac{1}{2}$ teaspoonfl | 1 teaspoonfl      |
| Glycerol, 10%                               |                     | single |                          | 1-2 g/kg          |
| Acetazolamide, 0 25 g                       | tablet              | single | $\frac{1}{5}$ tbl        | $\frac{1}{4}$ tbl |
| Lasix                                       | solution            | daily  |                          |                   |
| Mannitol                                    | solution            | single |                          | 1 g/kg            |
| Urea  | solution            | single |                          | 1 g/kg            |
| Novurit                                     | solution            | single |                          | 0 1 ml            |

Continued

| Age                |                   |                      | Administration                           | Notes   |
|--------------------|-------------------|----------------------|--|---|
| 1-3 years          | 4-7 years         | above 7 years        |  |   |
| $\frac{1}{4}$ tbl  | $\frac{1}{2}$ tbl | 1 tbl                | per os, 2-3 times a day                  | for epilepsy and paroxysmal tachycardia   |
| 0.8-1.0 ml         | 1.0-2.0 ml        | 2.0-3.0 ml           | slow intravenous                         | dilute in 20 ml of isotonic NaCl soln (0.25 mg in 1 ml)                                 |
|                    | 0.5-2 ml          | 1-3 ml               | intramuscular, intravenous drip infusion | for paroxysmal tachycardia and fibrillation   |
| 0.3-0.5 ml         | 0.5-0.7 ml        | 0.7-1.0 ml           | intravenous, intramuscular, subcutaneous | for bronchial asthma, cross heart block, ineffective cardiac massage                    |
| $\frac{1}{3}$ tbl  | $\frac{1}{2}$ tbl | $\frac{1}{2}$ -1 tbl | sublingual                               | for bronchial asthma  |
| mg/kg (for 2 days) |                   |                      | per os, intravenous, intramuscular       | 3 times a day for digitalization, then 2 times a day, with pulse control                |
| 0.1-0.2 ml         | 0.3-0.4 ml        | 0.5-0.8 ml           | intravenous                              | administered in isotonic NaCl soln  |
| 0.1 ml             | 0.15-0.2 ml       | 0.2-0.3 ml           | slow intravenous                         | repeat infusion not sooner than in 4-5 hours  |
| 0.025 g            | 0.025-0.05 g      | 0.05-0.1 g           | per os                                   | for hypernatraemia  |
| 2 teaspsfls        | 2 tbl spnfls      | 2 tbl spnfls         | per os                                   | for brain oedema  |
|                    | $\frac{1}{3}$ tbl | $\frac{1}{2}$ tbl    | per os                                   | for brain oedema  |
| 3-5 mg/kg          |                   |                      | intramuscular, intravenous               | to decrease liquor formation  |
| (as dry substance) |                   |                      | intravenous                              | the dose can sometimes be increased to 10 mg/kg   |
| (as dry substance) |                   |                      | intravenous, intramuscular               | dilute to 15 or 30% solution, use for oedema or after previous administration of liquid |
| per year of age    |                   |                      |  | same  |
|                    |                   |                      |  | cool to room temperature  |

| Preparation                         | Form                 | Dose   |                  |              |
|-------------------------------------|----------------------|--------|------------------|--------------|
|                                     |                      |        | under<br>1 month | 6 months     |
| <i>XV Hormones</i>                  |                      |        |                  |              |
| ACTH                                | solution             | daily  | 10-20 units      |              |
| Hydrocortisone                      | solution             | daily  |                  |              |
| Dexamethasone                       | tablets,<br>solution | daily  | 0.5 mg           | 0.5-1.0 mg   |
| Insulin                             | solution             | daily  |                  |              |
| Insulin                             | solution             | single |                  | 1 unit per 4 |
| Cortisone                           | solution             | daily  | 0.025            | 0.05         |
| Nerobol                             | tablets              | daily  | 1-2 mg           | 1-2 mg       |
| Nerobolil                           | solution             | daily  |                  | 1 mg/kg      |
| Prednisolone                        | solution             | daily  |                  |              |
| Retabolil                           | solution             | weekly |                  |              |
| <i>XVI Analeptics and antidotes</i> |                      |        |                  |              |
| Bemegrade, 0.5%                     | solution             | single | 1.0 ml           | 1.0-2.0 ml   |
| Nalorphine, 0.5%                    | solution             | single | 0.2 mg/kg        |              |
| Proserine, 0.05%                    | solution             | single |                  | 0.1          |
| Unithiol, 5%                        | solution             | single |                  | 1.0 ml       |
| <i>XVII Haemostatics</i>            |                      |        |                  |              |
| Vikasol, 1%                         | solution             | single |                  | 0.1 ml       |
| Calcium chloride, 10%               | solution             | single |                  | 1.0 ml       |
| Epsilon-aminocaproic acid           | solution             | single | 30-50 ml         |              |
| <i>XVIII Miscellaneous</i>          |                      |        |                  |              |
| Heparin                             | solution             | daily  | 50-1000 U/kg     |              |
| Panangin                            | solution             | single | 0.1 ml           | 0.1-0.2 ml   |
| Trasylol                            | solution             | single | 5000 U           | 10 000 U     |

*Concluded*

| Age   |            |               | Administration  | Notes   |
|---|------------|---------------|---|---|
| 1-3 years                                   | 4-7 years  | above 7 years |   |   |
| 20-40 units                                 |            | 40-60 units   | intramuscular   | for bronchial asthma and various allergies  |
| 3-5 mg/kg                                   |            |               | intravenous, intramuscular                              | to 5-10 mg/kg for cardiac arrest  |
| 0.2 mg/kg                                   |            | 0.2-0.3 mg/kg | intravenous, per os                                     |   |
| 1-2 U/kg<br>g of dry sugar in dropper       |            |               | subcutaneous<br>intravenous<br>intramuscular            | for allergic diseases, rheumatism, Addison's disease  |
| 0.05-0.1                                    | 0.1-0.2    | 0.1-0.3       |   | same  |
| 2-3 mg<br>a month                           | 2-3 mg     | 5 mg          | per os<br>intramuscular<br>intramuscular<br>intravenous | for cardiac arrest the dose can be increased to 2-3 mg/kg   |
| 1-2 mg/kg                                   |            |               |   |   |
| 1 mg/kg                                     |            |               | intramuscular   |   |
| 2.0-5.0 ml                                  | 5.0-10 ml  | 10.0-20.0 ml  | intravenous   | for barbiturate overdose  |
| 2-3 mg                                      | 3-5 mg     | 5-10 mg       | subcutaneous<br>intramuscular<br>intravenous            | antagonist to morphine  |
| ml per year of age<br>per 10 kg body weight |            |               | same<br>intramuscular<br>subcutaneous                   | for poisoning with heavy metal salts  |
| per year of age<br>per year of age          |            |               | intramuscular<br>per os                                 | the dose can be increased 3-4 times for cardiac arrest  |
| 50-80 ml                                    |            | 100 ml        | intravenous drip  |   |
| 50-1000 U/kg                                |            |               | intravenous<br>subcutaneous                             |   |
| 0.5-1.0 ml                                  | 2.0-4.0 ml | 5.0-10.0 ml   | intravenous<br>with glucose                             | drip for myocarditis, hypokalaemia, can be administered with strophanthin or digitalis preparations |
| 10 000-20 000 U                             |            |               | intravenous drip  | the dose can be doubled if the enzyme content is high   |

## 2 Paediatric Normal Values

| Test                              | Age   | Range  |
|-----------------------------------|---|--|
| <i>Blood</i>                      |   |  |
| 1 Total protein                   | under 1 m<br>2-6 m<br>6-12 m<br>1-4 y<br>5-14 y | 4 1-5 5 g% (41-55 g/l)<br>4 7-5 9 g% (47-59 g/l)<br>5 4-6 8 g% (51-68 g/l)<br>5 9-7 9 g% (59-79 g/l)<br>6 2-8 2 g% (62-82 g/l) |
| 2 Albumin                         | under 1 m<br>1 m-1 y<br>1-3 y<br>3-14 y         | 3 1-4 4 g% (31-44 g/l)<br>3 6-4 9 g% (36-49 g/l)<br>4 07-5 03 g% (40 7-50 3 g/l)<br>3 72-5 50 g% (37 2-55 g/l)                 |
| 3 Globulins                       | under 1 m<br>1 m-1 y                            | 1 6-2 6 g% (16-26 g/l)<br>1 6-2 9 g% (16-29 g/l)   |
| α <sub>1</sub> -globulins         | 1-2 y<br>3-14 y                                 | 0 15-0 35 g% (1 5-3 5 g/l)<br>0 12-0 30 g% (1 2-3 0 g/l)   |
| α <sub>2</sub> -globulins         | 1-2 y<br>3-14 y                                 | 0 41-0 66 g% (4 1-6 0 g/l)<br>0 35-0 95 g% (3 5-9 5 g/l)   |
| β-globulins                       | 1-2 y<br>3-14 y                                 | 0 52-0 83 g% (5 2-8 3 g/l)<br>0 40-0 92 g% (4-9 2 g/l)   |
| γ-globulins                       | 1-2 y<br>3-14 y                                 | 0 45-1 66 g% (4 5-16 0 g/l)<br>0 53-1 2 g% (5 3-12 g/l)  |
| 4 Bilirubin total                 | 1 m-14 y  | 0 2-1 2 mg% (3-20 μmole/l)   |
| 5 Bilirubin, direct (glucuronide) | 1 m-14 y  | 0 05-0 25 mg% (0 85-3 4 μmole/l)   |
| 6 Bilirubin, indirect (free)      | 1 m-14 y  | 0 15-1 0 mg% (2 57-17 1 μmole/l)   |
| 7 Glucose                         | 0-7 d<br>8 d-1 m<br>2 m-14 y                    | 30-75 mg% (1 7-4 2 mmole/l)<br>45-85 mg% (2 5-4 7 mmole/l)<br>60-100 mg% (33-55 mmole/l)                                       |
| 8 Lipids, total                   | 0-7 d<br>1 m-1 y<br>1-15 y                      | 170-450 mg% (1 7-4 5 g/l)<br>240-700 mg% (2 4-7 0 g/l)<br>450-700 mg% (4 5-7 0 g/l)  |
| 9 Uric acid                       | under 1 m<br>1 m-1 y<br>1-15 y                  | 2 4-5 0 mg% (1 4-2 9 mmole/l)<br>1-3 5 mg% (0 59-2 06 mmole/l)<br>1-4 0 mg% (0 59-2 36 mmole/l)                                |
| 10 Urea                           | under 1 m<br>1 m-1 y<br>1-14 y                  | 15-27 mg% (2 5-4 5 mmole/l)<br>15-35 mg% (2 5-5 8 mmole/l)<br>15-40 mg% (2 5-6 7 mmole/l)                                      |
| 11 Creatinine                     | 1-14 y  | 0 4-1 0 mg% (35-84 μmole/l)  |
| 12 Cholesterol                    | under 1 m<br>1 m-1 y<br>1-14 y                  | 60-115 mg% (1 6-3 0 mmole/l)<br>70-190 mg% (1 8-4 9 mmole/l)<br>140-250 mg% (3 7-6 5 mmole/l)                                  |
| 13 β-Lipoproteins                 | 1 m-14 y  | 300-600 mg% (3-6 g/l)  |
| <i>Blood Enzymes</i>              |   |  |
| 14 Alanine amino-transferase      | 1 m-14 y  | 0-40 U   |
| 15 Alkaline phosphatase           | under 1 m<br>1-2 m<br>3-5 m<br>6-11 m<br>1-14 y | to 59 U<br>40-156 U<br>34-162 U<br>34-140 U<br>38-138 U  |
| 16 Amylase                        | 1 m-14 y  | 16-30 U  |

Continued

| Test  | Age     | Range               |
|---|---------|---------------------|
| <i>Sedimentation Tests</i>                              |         |                     |
| 17 Thymol turbidity test                                | 0-14 y  | 0-4 U               |
| 18 Mercury test   | 0-14 y  | 80-100 U            |
| <i>Acid-base Balance and Gases of Blood</i>             |         |                     |
| 19 pH   | 1 m-1 y | 7.38-7.51           |
| 20 PaCO <sub>2</sub> (CO <sub>2</sub> partial pressure) | 1 m-1 y | 26.5-36.5 mm Hg     |
|   | 1-7 y   | 26.5-36.0 mm Hg     |
|   | 8-14 y  | 28-42 mm Hg         |
|   | 0-14 y  | 60-90 mm Hg         |
| 21 PaO <sub>2</sub> (oxygen partial pressure)           | 0-14 y  | 60-90 mm Hg         |
| 22 Bicarbonate, Standard (Sb)                           | 1 m-1 y | 18.5-25 mmole/l     |
|   | 1-4 y   | 18.5-24 mmole/l     |
|   | 5-7 y   | 20.5-26 mmole/l     |
|   | 8-15 y  | 20.0-26 mmole/l     |
| 23 Base excess or deficit                               | 1 m-1 y | (+2)-(-5) mmole/l   |
|   | 1-4 y   | (+1)-(-3.3) mmole/l |
|   | 5-7 y   | (+2.9)-(-4) mmole/l |
|   | 8-15 y  | (+2)-(-2.4) mmole/l |

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